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NERVOUS AND MENTAL DISEASES

FOR STUDENTS AND PRACTITIONERS

BY

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PREFACE TO THIRD EDITION

In revising the manual the author has made every effort to bring it up to date and profit by the criticisms of former editions, to the end that it may continue to serve as a convenient guide for the student and general practitioner in the closely related subjects of nervous and mental diseases. A description of tic embodying the present-day view of that disorder, and short descriptions of myotonia atrophica, progressive lenticular degeneration, and dysbasia lordotica deformans have been added. The chapter on general symptomatology and methods of examination has been amplified. Dementia paralytica instead of being classified under mental diseases has been described in the chapter devoted to diseases of the brain and cord. The importance of the examination of the cerebrospinal fluid and determination of the existence of the Wassermann reaction in the diagnosis of certain diseases of the nervous system has been realized and the latest views on these matters incorporated. Several new illustrations have been added. The section on mental diseases is of necessity brief, but it is believed will give the student a working knowledge of the intricate subject of which it treats. No attempt has been made to discuss either the psychology of these disorders or psychological methods of studying them, for the reason that although it is appreciated that further progress will be along these lines, the subject is still in a more or

less experimental stage. Reference, however, has been made to the theories of Freud in the description of hysteria. It is also recognized that many so-called functional diseases of the nervous system are really mental diseases. These, however, have not been described under that heading, insanity and idiocy alone being there considered. Reference, however, has been made to the fact that the functional diseases above mentioned are in reality mental diseases.

The author would acknowledge his obligation to the authors referred to in the text, as well as to those who have been mentioned in the prefaces of former editions, and in addition to the *Outlines of Psychiatry*, by White. He would also express his appreciation of the suggestions of Dr. Max Mailhouse, of New Haven, Conn.

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NERVOUS DISEASES

CHAPTER I

INTRODUCTORY OR HISTOLOGY

THE NEURONE

Constituents of the Neurone.—The nervous system is made up of a vast number of *units*, termed *neurones*. Each neurone is essentially of the same structure, and consists of a *cell body*, containing a nucleus, which in turn contains a nucleolus; from this cell body arise one or more *processes*, usually the latter, termed *dendrites;* and a process which differs in structure and function from these, called the axis cylinder process or *axone* (Fig. 1).

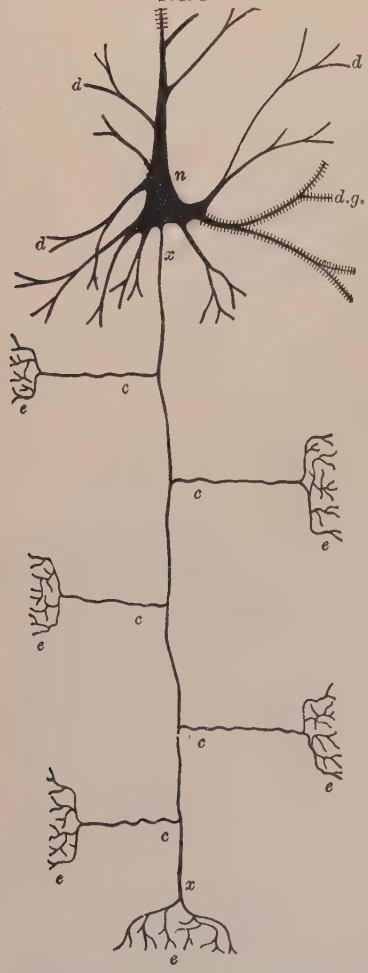
The **cell body** is composed of protoplasm embedded in a network of fibers. In most cells are found in this network granular masses which stain deeply, known as the *chromophilic granules* or Nissl bodies.

The **dendrites** are composed of protoplasm similar to that of the cell body, and are really part of it. They are broad and thick at their origin, and become narrower as they divide and subdivide. Upon them are a number of small, lateral projections termed *gemmules*. Within them may be seen fine filaments which pass into the cell body and through it into the other dendrites or the axone. These are the neuro-fibrils,[1] which were first described by Apathy.

The **axone** arises either from the cell body or a dendrite,

[1] For a fuller description see The Neurones, by Barker, Journal of the American Medical Association, March 31, 1906.

FIG. 1



Neurone with long axone proceeding as an axis cylinder of a nerve fiber: *n*, nerve cell proper; *d*, dendrites; *x*, axone; *d.g*, dendrite showing gemmulæ; *adg*, apical dendrite with gemmulæ; *c*, collaterals; *e*, end tufts. Pyramidal cell of the cerebral cortex. (S. Ramon y Cajal.)

usually the former. It differs structurally from the cell body and dendrite, being composed of *fine threads* embedded in a clear substance, which is probably protoplasm. Its surface is smooth, and the caliber is maintained without apparent diminution for some distance from the cell body. The length of an axone varies from a fraction of a millimeter to nearly 100 cm. (some fibers of the pyramidal tract). The short axones divide into a great number of branches; cells with axones of this type have been termed *dendraxones* or Golgi cells of the second type (Fig. 3). The long axones at intervals give off branches at right angles to their course termed *collaterals*. This type of cell is called an *inaxone* or Golgi cell of the first type. The axones and collaterals end by splitting into a brush-like arrangement termed the *end brush* or end tufts (Fig. 1). The exact way in which one neurone is connected with another is in dispute. The fibrils of the end brush or tufts may enter the dendrites of a

neighboring neurone; they may end in a free termination which only touches these dendrites, or they may end on the surface of other cell bodies in the terminal buttons of Auerbach, or as they are also called the end feet of Held (Fig. 2). The collaterals end similarly. Those axones which reach the muscles end in the so-called "motorial end plates or telodendrions." Axones and their collaterals, excepting for a short distance from their endings, are enveloped in a medullary sheath.

FIG. 2

Two large funicular cells of the spinal cord of the adult rabbit: *A*, section through a cell showing terminal buttons of Auerbach ending on the surface of the cell and on the dendrites; *B*, terminal buttons shown on the surface of a cell. (S. Ramon y Cajal.)

Functions.—The *cell body* may either originate efferent nerve impulses of various sorts (cortical cells); modify impulses received from another neurone (ganglion cells of cord); or receive and recognize afferent or sensory impressions.

Efferent impulses are carried away from the cell body by the *axone,* which may end either by surrounding with

its end brushes the dendrites of another cell or in the "terminals" of the muscles as described on p. 19 (Fig. 4).

Sensory impressions are collected either from the periphery (sensory end organs) or from the axone of another neurone by the *dendrites*. These latter probably also collect nutrient materials from the blood for the cell body. In addition to the neurone contiguous to the axone, each neurone is brought into relation with a number of other neurones by means of the *collaterals*.

Fig. 3

Neurone with short axone immediately breaking up into numerous fine filaments: *nc*, nerve cell proper; *c*, axone; *d*, dendrites. From the cerebellum. (Andriezen.)

The *life* of the processes is dependent upon the cell body; if this is destroyed they degenerate and die; or if part of a process is cut off from the cell body by any reason, it dies.[1]

Neurones: Their Relations to Each Other.—The *cells* are found in the gray matter of the brain, cord, and ganglia of the peripheral nerves. A group of them which together

[1] In such an event the entire neurone suffers, but not so rapidly nor to such an extent as the separated part.

control some particular function of the body is known as a *centre;* for instance, that collection of cells situated in the lower part of the central convolutions which controls the movement of the tongue is the tongue centre.

FIG. 4

Illustrating the varying relations of neurones to each other. *NC*, nerve cell; *D*, dendrites; *x*, axones; *C*, collaterals; *E*. end brush; *M*, muscle fiber. (After Obersteiner.)

TRACTS

Projection Fibers.—The *axones* form the various tracts, and are found principally in the peripheral nerves and white matter of the brain and cord. Those fibers which connect cortical centres directly with cells in the basal ganglia, the nuclei in the pons and medulla and in the gray matter of the

cord are termed *projection fibers* (Fig. 5). These fibers form the pathways by which motor impulses are carried from the cortex to the spinal centres, and by which sensory impressions are brought from certain ganglionic masses in the medulla and base of the brain to the cerebral cortex.

Fig. 5

The projection tracts joining the cortex with lower nerve centres. Sagittal section, showing the arrangements of tracts in the internal capsule: *A*, tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side; *B*, motor tracts from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at *K*; *C*, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; *D*, visual tract from the optic thalamus (*OT*) to the occipital lobe; *E*, auditory tract from the internal geniculate body (to which a tract passes, from the *VIII N* nucleus, *J*) to the temporal lobe; *F*, superior cerebellar peduncle; *G*, middle cerebellar peduncle; *H*, inferior cerebellar peduncle; *CN*, caudate nucleus; *CQ*, corpora quadrigemina; *Vt*, fourth ventricle. The numerals refer to the cranial nerves.

These pathways, together with certain columns in the spinal cord and the peripheral nerves, form the *motor* and *sensory tracts*, which are therefore composed of two or more neurones. (See pp. 23, 25.)

Association Tracts and Association Centres.—All that part of the cortex (comprising about two-thirds) not con-

nected with projection fibers—*i. e.*, that part in which are *not* included the sensory and motor regions and special sense centres—contains, according to Flechsig, centres in which the various sensory impressions are collected, arranged, and coördinated. These centres are termed by him *association centres*, and are connected with the sensory and motor regions and special-sense centres by collections of fibers termed *association tracts* (Fig. 6).

Fibers also connect the centres of one side with the corresponding ones in the other. These are termed *commissural fibers.*

The location of the *cerebral centres* will be given under Cerebral Localization.

FIG. 6

The association fibers: *A*, between adjacent convolutions; *B*, between frontal and occipital areas; *C*, between frontal and temporal areas, cingulum; *D*, between frontal and temporal areas, fasciculus uncinatus; *E*, between occipital and temporal areas, fasciculus longitudinalis inferior; *CN*, caudate nucleus; *OT*, optic thalamus.

The Motor Tract.—The motor tract has its origin in the centres situated in the ascending frontal convolution (Fig. 84). The axones of these cells (Fig. 7) converge as they pass down through the brain until they reach the internal capsule, where they form a compact bundle occupying most of the posterior limb. They pass hence through the crus, pons, and medulla, and in the lower part of the latter most of them

cross to the opposite side. Previous to this, however, in the lower part of the pons and the upper part of the medulla, fibers have been given off, which decussate and pass to the

Fig. 7

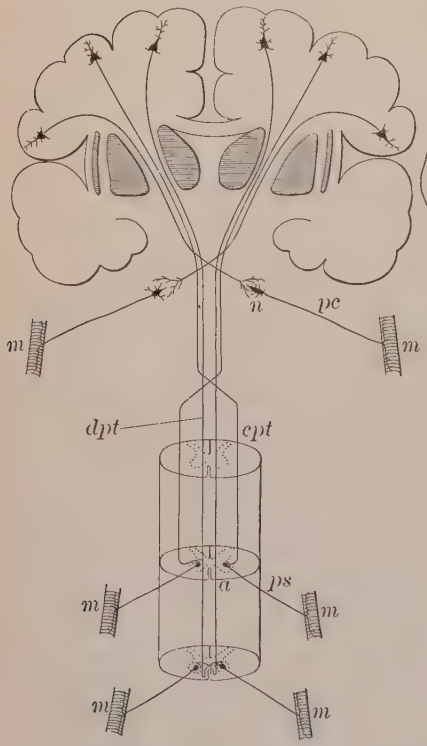


Fig. 8

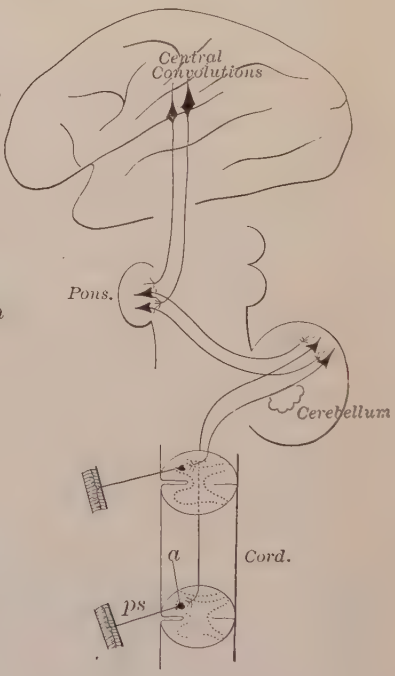


Fig. 7.—Diagram of the direct or voluntary motor tract, showing the centre of the motor impulses from the cerebral cortex to the voluntary muscles: *m*, muscles; *n*, cells of nuclei of motor cranial nerves in pons and medulla; *a*, motor cells in anterior horns of spinal cord; *dpt*, direct pyramidal tract; *cpt*, crossed pyramidal tract; *pc*, peripheral cranial nerve; *ps*, peripheral spinal nerve. (Van Gehuchten.)

Fig. 8.—Diagram of the indirect or involuntary motor tract.

nuclei of the motor cranial nerves (Fig. 7). Those fibers which cross in the lower part of the medulla pass down in the posterior part of the lateral columns of the cord, as the

crossed pyramidal tract, and the "terminals" of these fibers surround the cells in the anterior horns. The axones from *these* cells and from the cells of the motor cranial nerve nuclei, mentioned above, form the motor division of the peripheral nerves.

The comparatively few fibers which have *not* crossed pass down in the middle portion of the anterior columns, forming the *direct pyramidal tract* or column of Türck, the fibers of which cross in the anterior commissure at different levels and also connect with the motor cells in the anterior horns (Fig. 7).

Function.—This *motor tract* is concerned in all voluntary muscular movements. The cortical cells originate the impulses and control or inhibit the functions of the spinal cells. The cord cells receive and distribute the motor impulses to the muscles, maintain their nutrition, and with the sensory cells form reflex centres.

There are also *indirect motor paths* which run from the cortex to the thalamus, and red nucleus. From the red nucleus connection is had with the pons and cerebellum, from whence fibers pass down the lateral columns (rubrospinal and anterior descending cerebellospinal tracts) to communicate with the anterior horn cells and through the peripheral nerves the muscles (Fig. 8). Their function is to play a part in coördinating muscular movements and maintaining muscle tone.

The Sensory Tract.—The sensory tracts are much more complicated and their course not so well known as the motor. There are probably different pathways for the various *forms* of *sensation*, viz., tactile, pain, temperature, and muscle. The cells of the first neurone are situated in ganglia upon the posterior nerve roots or sensory cranial nerves, as the case may be. Each of these cells gives off a long process, a dendrite which runs to the periphery as part of a peripheral nerve, and ends, according to the peculiar form of sensation that it conducts, in one of the various specialized *end organs* in either the skin, muscles, joints, or organs of special sense. From the cell the *axone* also arises, enters the spinal cord as part of a posterior root, and divides

into a long ascending and a short descending branch. Some of the former pass up the cord in the posterior columns in the

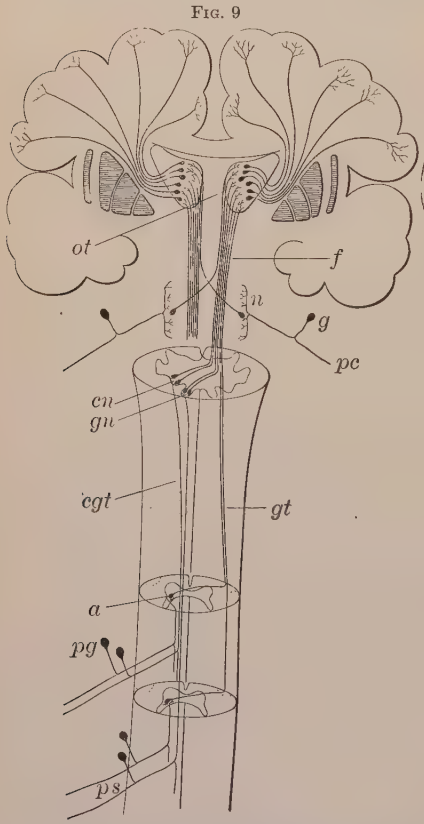


Fig. 9

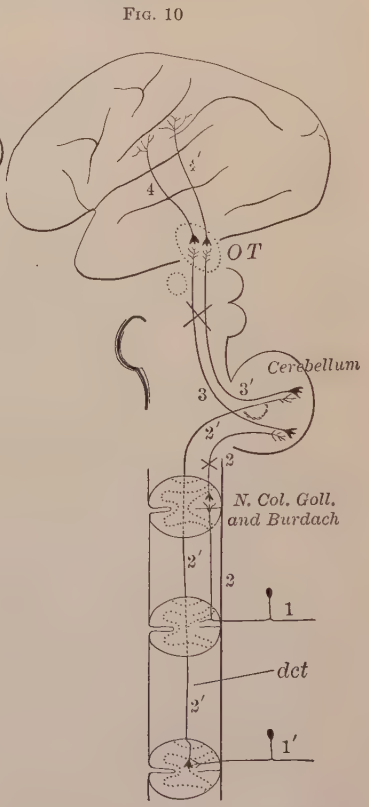


Fig. 10

Fig. 9.—The direct sensory tract: *ps*, peripheral spinal nerves; *pg*, ganglion on posterior roots of spinal nerves; *gt*, Gowers' tract; *cgt*, columns of Goll and Burdach; *cn*, nucleus cuneatus; *gn*, nucleus gracilis; *a*, cells in posterior horn; *pc*, peripheral cranial nerve; *g*, ganglion on cranial sensory nerve; *n*, cells of cranial sensory nerves in medulla; *f*, fillet; *ot*, optic thalamus.

Fig. 10.—Indirect sensory tracts: *dct*, direct cerebellar tract. The numbers represent the different series of neurones.

nuclei of which they end. The cells of these nuclei are the beginning of another neurone, the axones of which form the

lemniscus or fillet, which also receives fibers from the sensory cranial nerve nuclei (Fig. 9). Here they decussate and the fibers run to cells in the optic thalamus, axones from the cells of which, after passing through the posterior part of the internal capsule, reach the cortex, where they are brought into relation with the motor neurones. Sensations of **touch** are probably conducted by this path.

Other fibers leave the nuclei of the posterior columns, and by means of the inferior cerebellar peduncles of the same side reach the cerebellum. From these another series of fibers, by way of the superior peduncles, passes to the optic thalamus and red nucleus, and from these by other neurones the tract reaches the cortex. Sensations from the **muscles** upon which the appreciation of the direction of motion of the limbs depends and **joints**, by which we recognize the position of the limbs, both of which have to do with coördination, probably travel by this route (Fig. 10). (See also Clarke's Column.)

Another series of fibers, after entering the cord, surrounds, with its terminals, cells in the posterior horns of the gray matter. From these cells axones arise which pass to the opposite side, where they form the *anterolateral* or *Gowers' tract* (Fig. 9). Part of the fibers of this column, probably by way of the fillet, reach the optic thalamus and corpora quadrigemina, from which points their course is doubtful; while others by way of the superior peduncles go to the cerebellum. Sensations of **pain** and **temperature** are probably conducted by this tract.

Still other fibers join the cells of *Clarke's column*, the axones of which form the *direct cerebellar tract*, which, without decussation, ends in the cerebellum through the inferior peduncle. From hence fibers pass to the red nucleus and optic thalamus, the cells of which give off axones which pass to the brain cortex (Fig. 10). This tract also conducts sensations from the *muscles* and *joints*.

Collaterals from the sensory fibers pass into the gray matter, where they join the motor and sensory cells. By means of these and the short descending branches the different levels and various sensory tracts are brought into relation.

The **special sense tracts** will be described in connection with the diseases of their peripheral nerves.

In addition to the cells and their processes just described the nervous system is composed of connective tissue, neuroglia, bloodvessels, and lymphatics, the whole being enveloped in the membranes which constitute the meninges.

CHAPTER II

GENERAL PATHOLOGY

The functions of the nervous elements may be *impaired* by disease or abnormality of any of their structures.

INFLAMMATION

In the nervous system, as elsewhere, **inflammation** is the reaction of the organism to an irritant. This irritant may be the product of microbic action or tissue change, or of some chemical substance, as lead, alcohol, arsenic, etc. This inflammation may be *simple*, without exudation or destruction of tissue; or there may be exudation, and destruction of nerve tissue, with the formation of connective tissue or pus; furthermore, the process may be chronic or acute.

The following table (from Dana) shows the various **forms** and their **causation**:

Classification of Inflammations

Form.	Cause.	Example.
Simple exudative, with or without necrosis.	Microbic or toxic.	Meningitis; poliomyelitis.
Purulent, with or without necrosis.	Microbic or necrobic.	Meningitis and encephalitis; acute purulent myelitis.
Productive or proliferative.	Microbic or toxic.	Chronic meningitis; leprous neuritis.

DEGENERATION AND SCLEROSES

By **degeneration** we mean a gradual death of the nerve cells and their processes. This may be acute, chronic, primary, or secondary.

Acute degeneration is due to the cutting off of the blood supply, to traumatism, and to the effects of inflammation. It produces the condition known as *necrosis* or *softening*.

Chronic degeneration is caused by the continued action of various poisons, especially metallic and those of infectious diseases, and is accompanied by the formation of connective tissue and consequent sclerosis.[1]

A **primary degeneration** is one due either to some inherent defect, as in hereditary ataxia; or to the direct action of some poison, as in some cases of lateral sclerosis and progressive muscular atrophy.

A **secondary degeneration** is due either to the cutting off of a nerve fiber from its trophic centre—*i. e.*, the *cell body*—as the degeneration of the pyramidal tract following a hemorrhage into the internal capsule; or to a deficiency in the blood supply (p. 333). Primary and secondary degeneration may occur in the same disease. When a peripheral nerve is injured sufficiently to rupture the sheath of Schwann or neurilemma (for diagram of normal nerve see Fig. 11) the white substance of Schwann or medullary sheath is driven away from the point of pressure in both directions and the axis cylinder is disintegrated and mingled with it. In a short time degeneration occurs in the nerve on both sides of the point of injury. On the central side it is limited to the neighborhood of the injured spot; on the peripheral side the changes extend to the termination of the nerve. First, the medullary sheath breaks into segments, then into smaller masses and drops (Fig. 12), until, finally, a granular mass alone remains, and in some cases this may become absorbed, leaving an empty sheath of Schwann. The axis cylinder in mild cases may remain intact and resume its functions when the medullary sheath is regenerated. In other cases the axis cylinder becomes broken up and disintegrated. At this time numerous nuclei develop and line the neurilemma. If regeneration does not occur these finally disappear and nothing is left but a strand of connective tissue.[2]

[1] In this progress it is probable that there is also some increase of the neuroglia.

[2] Slight changes may be found in the cells (reaction at a distance) and nerve fibers extending to them.

Different views are held as to the method of regeneration. Many believe that it is due to a new outgrowth from the

FIG. 11

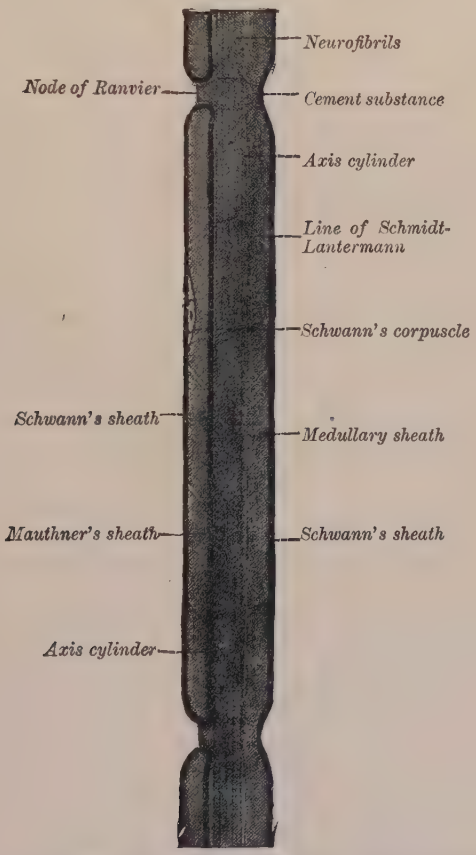


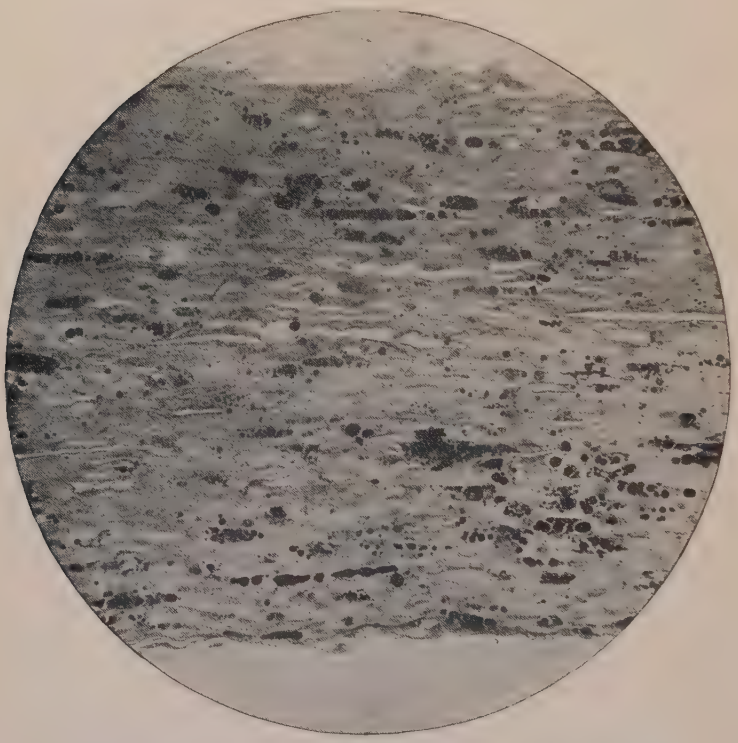
Diagram of the structure of a medullated nerve fiber, showing two different views concerning the relations of the sheaths of Mauthner and of Schwann. Compare the right and left sides. (Szymonowicz.)

central stump, others, notably Ballance and Stewart[1] believe that the new axone is formed in the peripheral end by the

[1] Healing of Nerves.

activity of the neurilemma cells which proliferate and form chains of overlapping spindle cells from which, as soon as they unite with the central end, nerve fibers are developed.

Fig. 12



Degeneration of a nerve fourteen days after injury. Osmic acid stains the fatty debris of the medullary sheath black.

Gliosis.—When we have an extensive proliferation of neuroglia the process is termed gliosis or gliomatosis. This process is found in syringomyelia.

General Considerations.—It must be remembered that nerve cells destroyed do not develop again. The nerve fibers in the brain and cord, if destroyed, are not regenerated,

but a peripheral nerve fiber may be destroyed, and if its trophic centre is intact it will grow again. If the fibers are divided it is essential that the ends be brought into contact by suture.

Other **forms of disease** which may affect the nervous elements are: malformations, incomplete or defective development; hyperemia; anemia; edema; diseases of the bloodvessels; hemorrhage; thrombosis or embolism; atrophy softening; syphilis and tuberculosis; tumors; parasites; functional disorders; disorders associated with glandular defects, especially the ductless glands; and disorders of nutrition and metabolism.

CHAPTER III

GENERAL SYMPTOMATOLOGY AND METHODS OF EXAMINATION

THE **symptoms** caused when a nerve centre or tract is affected by any of the lesions mentioned in the preceding chapter depend upon whether the lesion is destructive or irritative. If destructive, the function of the involved part is diminished or destroyed; if irritative, its function is increased. Symptoms produced by a combination of these two kinds of lesions may be present; for instance, a cerebral tumor, while it causes destruction of the nerve elements directly affected, will cause irritation of the *neighboring parts*. A lesion which may at first be irritative may finally be destructive. For instance, a meningitis may at first cause increased cerebral action with convulsions, etc.; but, finally, as the disease progresses, these parts are destroyed and coma and paralysis result.

Symptoms resembling those produced by an irritative lesion may also be produced if the higher or inhibitory centres in the brain are destroyed, thus allowing overaction of the lower or spinal centres to occur. (See Contracture.)

Neurosis and Psychosis.—The general term for any derangement of the nervous system, *exclusive* of those of the higher or mental functions, is *neurosis*. This term is more commonly applied, however, to disorders of a *functional nature*, or those in which we can find no *apparent organic cause*.

A disorded mental state is known as a *psychosis*.

The following **prefixes** are used to indicate derangements of function of the nervous system, viz., "hyper," meaning excess; "hypo" or "hyp," which means diminution; "a" or "an," which indicates entire loss; and "para," meaning perversion. For example, we speak of *hyperesthesia*, meaning

increased sensibility; *hypesthesia,* meaning diminished but not loss of tactile sensibility; *anesthesia,* or loss of tactile sensibility; and *paresthesia,* for perverted sensibility.

SYMPTOMS CAUSED BY DISORDERS OF THE HIGHER OR PSYCHICAL CENTRES

These are mostly found in mental diseases, as insanity, idiocy, and imbecility, and will be discussed under that head. We may, however, find loss of will; failure of memory; lack of control; inability to concentrate the attention; mental excitement; or depression in patients who are not insane. (See pp. 444 to 455.)

Consciousness may be considered one of the higher functions of the brain. Impairment or loss of it is an important symptom of many nervous diseases both organic and functional. This loss may be due to disease of the brain cortex or to the inhibitory action of lesions elsewhere in the brain. For instance, the loss of consciousness in cerebral hemorrhage is due probably to an irritative inhibitory action on the cortex.

Stupor and Coma.—If the patient can be partially aroused by ordinary stimuli, we call the condition *stupor;* if he cannot be aroused, we term it *coma.*

In *stupor* the reflexes are active; in *coma* they are not, and the pupils are either contracted or dilated and immobile; the pulse slow, sometimes irregular or frequent; and the respiration slow and weaker than normal. The relaxation of the palate allows it to vibrate with the passing current of air, causing the so-called *stertorous breathing.* Frequently the respiration is of the Cheyne-Stokes type.

In *examining a case* of stupor or coma the head should be carefully looked over for possible injury; the existence of paralysis or spasm detected; the presence or absence of *reflexes,* superficial and deep, noted; the breath examined for the odor of alcohol or opium; and the urine for evidences of nephritis or diabetes. Furthermore, it must be ascertained whether the patient is an epileptic who has just had a con-

vulsion, or is a syphilitic or hysteric. The differential diagnosis of coma will be discussed under the head of Apoplexy.

SYMPTOMS CAUSED BY OVERACTION OF THE MOTOR TRACT

Convulsions.—A convulsion, as defined by Herter, consists of "involuntary, paroxysmal, purposeless muscular contractions of variable intensity and duration, and of extensive or limited distribution." Consciousness may be present or lost. Convulsions are also termed *spasms;* but this term is usually applied to those of local or limited distribution; as, for instance, the muscles supplied by one nerve, as the facial. Convulsions may be *tonic, i. e.*, when the contraction is slow and continuous; and clonic, *i. e.*, when the muscles rapidly and alternately contract and relax. A lesion limited to the centres of a small group of muscles may cause a general convulsion. In such cases there are, first, spasm of the muscles governed by the affected centres, and then a gradual involvement of all the muscles. (See Jacksonian Epilepsy.)

Contractures, Contractions, and Spasticity.—A *tonic* spasm of long duration causes a *contracture.* Such muscles are said to be *spastic* or in a state of *spasticity.* This may be due either to irritation of the motor tract; to a destructive lesion of the cortical motor neurones and consequent overaction of the spinal reflex arcs, due to the deprivation of the inhibitory influence of the former; or it may be of functional origin, as in hysteria. Normal muscle tone probably depends on the integrity of spinal reflexes and the influence of the cerebellum exerted through the indirect motor tracts (p. 25). Loss of any part of this mechanism causes hypotonicity of the muscles, while removal of the influence of cortical inhibition exerted through the pyramidal tract causes hypertonicity. The reason why in some cases of central lesion there is a flaccid palsy while in others most intense contractures develop is in doubt. A reasonable explanation is that when the direct pyramidal tract is

destroyed, motor impulses in time pass through the indirect or extra tracts, so that if the direct or inhibitory tract is destroyed and the indirect is intact the muscles become hypertonic and contracted. The normally stronger or flexor muscles overpowering the weaker or extensor muscles, thus causing the peculiar deformity known as a contracture (Fig. 60). If, however, both tracts are destroyed, no motor impulses can be sent to the muscles, tonicity is abolished, and hence a permanent flaccid palsy results. When there is an irritative lesion of the indirect tract with not much involvement of the direct, marked contractures with intermittent tonic spasms of different groups of muscles occur. These are different from athetoid movements[1] (see below; also p. 327).

A contracture long continued often causes actual organic shortening of the muscle, and the condition is then termed a *contraction*. An unequal paralysis of antagonistic muscles will also cause a contracture, often seen in poliomyelitis.

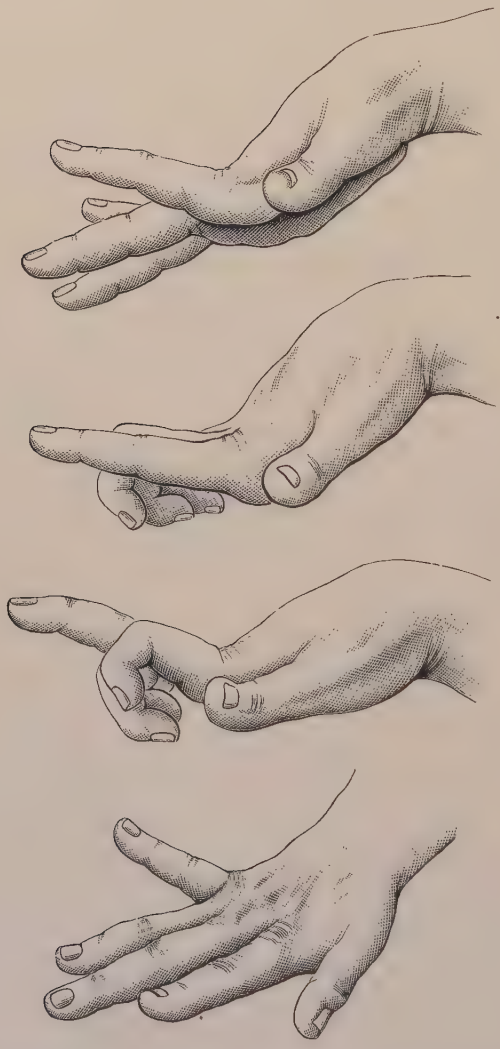
Examination.—Contractures can be discovered by passively flexing and extending the limb, when an abnormal resistance will be noted, the limb often bending like a piece of lead pipe; by supporting the proximal end of a limb and allowing the distal end to drop by its own weight; and by noting the deformity which is produced by the unequal spasm of antagonistic muscles, one group of muscles overpowering their antagonists (Fig. 60). Functional contractures, unless of very long duration, can be differentiated from those of organic cause by noticing that they disappear while under the influence of sleep or an anesthetic (p. 474).

Athetoid Movements or Athetosis.—Lesions in the optic thalamus[2] sometimes give rise to a peculiar form of clonic spasm which causes slow, irregular vermicular movements of the fingers, toes, and rarely of the face, which are termed athetoid movements. The movements are aggravated by voluntary motion and excitement. They are a sequel of

[1] Weisenburg, Univ. of Penna. Med. Bull., July and August, 1905, and Spiller, Amer. Jour. Med. Sci., June, 1910, p. 822.

[2] Lesions of the red nucleus, the tegmentum and vicinity have also been found.

Fig. 13



Examples of the position of the fingers in the movements of athetosis. (Strümpell.)

hemiplegia, particularly of the form that occurs in childhood. Often the paralysis has almost or entirely disappeared, and such cases may be mistaken for chorea. Lesions in the motor tract may cause tremor and choreiform movements (pp. 39, 41). A careful inquiry will usually elicit the history of a previous paralysis (Fig. 13).

Fibrillary Tremor.—A fibrillary tremor or contraction is an involuntary contraction of small numbers of muscle fibers. They are seen as fine wave-like contractions running along the muscle in which they occur. Movement of the part is not caused by them. They occur in muscles which are slowly atrophying from loss of neurotrophic influence. When they do not occur spontaneously they may be elicited by tapping the muscle with the finger or some other mechanical irritation. They are usually seen in chronic poliomyelitis, progressive spinal muscular atrophy, syringomyelia, and arthritic atrophy; they sometimes occur in neurasthenia, and may also be caused by exposure to cold and long-continued heat (p. 440).

Myoidema.—Myoidema is a phenomenon also occurring in slowly atrophying muscles due either to lower neurone or general wasting diseases. It is characterized by the formation of a ridge of nodules when the muscle is squeezed or a hard substance drawn across it. It is most frequently seen in the biceps and pectoral muscles.

Tremor.—By tremor is meant a to-and-fro movement of a part due to a more or less rapid involuntary and rhythmical contraction of antagonistic muscles. It may affect the muscles of the limbs, or neck, face, and tongue.

Tremor is probably due to either an interruption or exaggeration of the impulses as they pass from the cortex to the muscle.

Tremors may be rapid or coarse, and are divided into those which only occur during voluntary movement (intention tremors); those which are constant, but are increased by voluntary movement; and those which cease for a time or diminish after voluntary movement.

Examination.—If a tremor is not apparent it may be discovered either by making the patient extend the arms and

hands at full length, with the fingers separated, especially if a sheet of paper is laid across the hand, or by performing some voluntary act, as taking a glass of water. To be sure that a tremor is not an intention tremor, it is a good plan to place the patient in a recumbent position. Tremor of the tongue is tested by causing it to be protruded; that of the lips and face, by showing the teeth or whistling.

Care must be taken not to mistake a tremor of the head due to the neck muscles for shaking of the head due to tremor of the trunk.

Tremor is always a symptom, although at times it is exceedingly difficult to ascertain the cause. In studying such a case it should be noted if it is fine or coarse, rapid or slow, and if it varies in regularity or equality. The different conditions which may produce tremor must be borne in mind. With the exception of the tremor of paralysis agitans, all tremors in the early stages of the trouble are of the intention type; but as the disease progresses they may become constant. Tremors due to organic disease are usually slow; those of functional or toxic origin are usually rapid.

The following table gives the various **causes** of tremor and their character:

Cause.	Type of tremor.	Rapidity.
Toxic: Mercury, Arsenic, Lead, Alcohol, Tobacco, Tea, Coffee.	Intention in early stages; later may become constant; may then be increased by exertion.	Rapid.
Neuroses: Hysteria, Neurasthenia, Exophthalmic goitre.	Ibid.	That of hysteria sometimes is slow; others always rapid.
Senility—arteriosclerosis.	Ibid.	Slow.
Heredity.	Ibid.	Rapid.
Any disease of brain, spinal cord, or peripheral nerves, excepting multiple sclerosis and paralysis agitans.	Ibid.	May be slow or rapid, or both combined.
Paralysis agitans (see p. 422).	Often ceases for a few seconds after muscular exertion.	Slow.
Multiple sclerosis.	Intention only.	Slow, somewhat irregular.
Cerebrospinal syphilis.	Intention or constant.	May be slow or rapid.
Paresis.	Intention or constant.	May be slow or rapid.

Choreic Movements.—These are sudden, jerking, incoordinate, non-rhythmical, and non-purposive movements of different groups of muscles. They usually cease during voluntary effort. Tics, or the contractions of a group of muscles acting habitually together, as of the face, have been sometimes termed choreic movements; but they are more regular and coördinate than the true movements of chorea (p. 430).

Forced Movements.—These are movements in which the patient is forced against his will to move in a certain direction, to one side, forward or backward, or to rotate.

Associated movements are seen when a movement of a non-paralyzed limb causes a movement in the paralyzed one. Usually the movements in the paralyzed limb are limited in range and are best seen when the non-paralyzed limb is performing some fine coördinate movement, as buttoning the clothes; they may, however, be extensive in range.

SYMPTOMS DUE TO DESTRUCTIVE LESIONS OF THE MOTOR TRACT

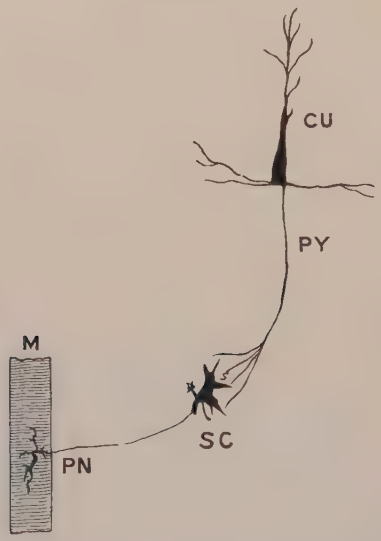
Paralysis.—The result of a destructive lesion, involving the motor tract, is termed *paralysis*. If the function is not entirely destroyed it is sometimes called *paresis*. When one limb or a single group of muscles of a limb is affected it is called *monoplegia*. When all or most of the muscles of one side of the body are paralyzed it is termed *hemiplegia*. Paralysis of the lower limbs is known as *paraplegia;* and as *diplegia* when it affects corresponding extremities, as two arms, two legs, or all four limbs. When one side of the face and the limbs of the opposite side are paralyzed we speak of an *alternate* or *crossed paralysis*. If abnormal muscular rigidity is present, the paralysis is said to be *spastic* (p. 36).

Varieties.—Paralyses are divided according to the portion of the motor tract affected, into *central*, in which some portion of the upper or central neurone is the seat of the lesion; and *peripheral*, in which some portion of the peripheral neurone is diseased (Fig. 14).

TABLE SHOWING DIFFERENCE BETWEEN CENTRAL AND PERIPHERAL PARALYSES

	Diseases of primary neurone or central.	Diseases of secondary neurone or peripheral.
Nutrition of muscles.	Good.	Poor; muscles atrophied.
Tone of muscles.	Increased; muscles usually rigid and spastic.	Diminished; muscles flaccid.
Electrical reaction. (See Electricity.)	Same as normal muscle.	Changed; either quantitative decrease or reaction of degeneration present.
Reflexes.	Increased.	Diminished or lost.

FIG. 14



CU represents a cell in the motor region of the brain cortex; *PY* is its axone, which forms part of the pyramidal tract; *SC* represents a cell in the gray matter of the cord (anterior horns); *PN*, its axone, forming part of a peripheral nerve; *M*, muscle. A lesion destroying *CU* or any part of the tract *PY* causes a central palsy; a lesion destroying *SC* or any part of the tract *PN*, a peripheral palsy.

Methods of Examination.—In examining a case of suspected paralysis it is important to remember that sometimes muscular weakness may be caused by exhaustion from some

acute illness or rachitis, and that the movements of a limb may be restricted, not from weakness due to lesion of the nervous system, but from pain caused by making the movement. This is often seen in joint inflammations. Impairment of motion may also be due to complete or partial ankylosis.

In most cases the existence of paralysis is obvious. The inability to use the part if an entire limb is paralyzed, or the resultant deformity when a group of muscles only is involved (due to the unbalanced action of antagonistic muscles; for example, the drooping of the eyelid in the paralysis of the motor oculi nerve), is at once apparent.

However, when the weakness is slight, or there is a question of diagnosis from the conditions above mentioned, certain *tests* are useful. In making these, several facts must be borne in mind, *i. e.*, in making a single muscular movement, as, for instance, extending the fingers, a number of muscles are brought into action in addition to those which actually do the work. Beevor[1] has divided the muscles used in making a movement into four classes: (1) Prime or principal movers. (2) Synergic muscles. (3) Fixation muscles. (4) Antagonists. The prime movers are not always all brought into action; thus in flexing the elbow, if the weight of the forearm is lifted only, the biceps contracts and the supinator longus does not; when there is resistance to the movement both muscles contract. The synergic and fixation muscles prevent movements taking place that are not desired. For instance, in supinating the forearm the biceps is the principal muscle; it also is a flexor of the forearm When, however, supination is the movement desired, the triceps contracts and prevents flexion; the triceps then is a synergic muscle. If the fingers are flexed the extensors must also contract, otherwise flexion of the wrist will occur; the extensors, therefore, are fixation muscles. Weakness of groups two and three may cause an apparent weakness of group one. This is frequently seen when in paralysis of the extensors of the

[1] The Coördination of Single Muscular Movements in the Central Nervous System, Jour. Amer. Med. Assoc., July 11, 1908, p. 90.

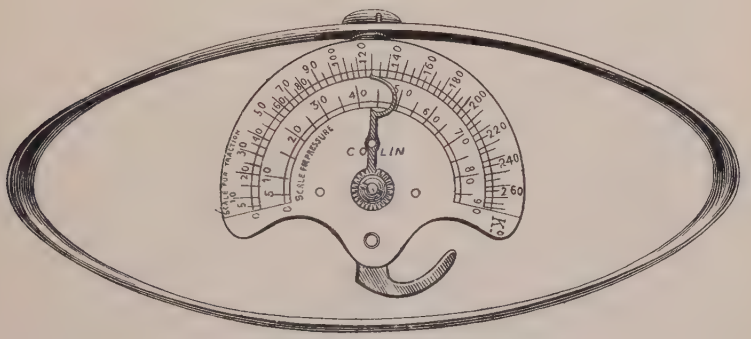
fingers there is apparent weakness of the flexors shown by a weak grip due to the hand going into flexion; but if the wrist is supported and flexion prevented, the strength of the grasp becomes normal. The antagonists are muscles which produce exactly the opposite action, as the biceps and triceps. In making a movement the antagonists should relax; thus in extending the forearm on the arm the biceps, supinator longus, and brachialis anticus should relax; this may be of use in diagnosis (p. 471). In examining the patient to detect weakness we cause the patient to use the suspected muscles, comparing the movements with those of the opposite side. For instance, if the muscles of the *face* are being examined we make the patient wrinkle the forehead, open and close the eyelids, smile, whistle, and draw the mouth from side to side. Weakness of the muscles of mastication may be detected by causing the patient to bring the jaws together firmly, while the fingers are held lightly over the masseter and temporal muscles of each side. Differences in the amount of contraction or absence of it can thus be detected. The external pterygoids are tested by asking the patient to move the lower jaw from side to side. Weakness is shown by inability to move it toward the normal side and by deviation of the point of the chin toward the weak side when the mouth is opened widely. Weakness of the internal pterygoids is shown by inability to push the lower jaw forward.

Paralysis of the *soft palate* can be detected by causing the patient to open the mouth and make the sound "ah." If both sides are paralyzed the palate will be seen to move but slightly or not at all. If only one side is paralyzed the palate will be drawn upward on the sound side only.

Weakness of the *tongue* is detected by noticing if it is protruded with difficulty or to one side; and if when protruded it cannot be kept so long. Weakness of the muscles which move the *eyeball* may be roughly tested by covering one eye and causing the patient to follow the finger while it is moved in various directions, the head, of course, being kept fixed. Associated movements of the eyes, laterally and up and down, should also be tested. Slight weakness may

only be detected by employing delicate tests and apparatus, for which the patient had best be referred to an ophthalmologist. When such weakness is present the patient may also complain of seeing double, or diplopia (p. 56). To test the movements of the *iris*, we notice if it contracts when a bright light is brought before the eye and dilates when the light is removed, and if it dilates when looking at a distance and contracts when looking at a near object, as, for example, the end of the finger brought toward the patient's nose. The eye being tested must be kept covered.

Fig. 15



Hand dynamometer.

The *arms* are examined by causing the patient to make the various movements while counterforce is applied by the examiner. Thus to test the biceps, the forearm is flexed on the arm, while the examiner grasps the wrist and tries to prevent it. The grasp of the hand is determined by having him squeeze your hands with his; or by using the dynamometer (Fig. 15) and comparing the records made. If a *leg* is weak, it may be dragged as the patient walks. Similar methods are used in examining the legs, as for the arms, the various movements of the limb being attempted, and if the weakness is not apparent, doing the same against the resistance of the examiner. In slight cases of paresis of the extensor muscles of the foot, examination of the soles of

the shoes may be of service, as in such cases the toes are worn off unduly.

If *coma* is present, we can often detect paralysis by noticing the less resistance with which the limb falls when allowed to drop as compared with the other side.

Atrophy, if marked, is at once apparent. Slight degrees are detected by noticing that the muscles are more flaccid than normal and by *measuring*. To measure we select a fixed point, as the anterior iliac spine, in the case of the leg, from which, at equal distances on each side, we mark a number of points; at these points we take the circumference of the limbs and compare the results. Valuable information is also obtained by using the electrical current. (See Electricity.)

SYMPTOMS DUE TO IRRITATIVE LESIONS OF THE SENSORY TRACT

Hyperesthesia.—Irritation of the sensory tract may cause increased sensibility to pain (increased sensibility to *other* forms of sensation is not yet capable of being detected), a condition often termed *hyperesthesia*, but more properly known as *hyperalgesia*. When it exists, slighter stimuli than normal should cause painful or disagreeable sensations. Irritation of any portion of the sensory tract may produce it.

Pain.—This is another expression of sensory tract irritation. At times its character and situation may be of diagnostic value. It may be limited to the distribution of a certain nerve, as in neuralgia and neuritis; it may be diffuse, as in cerebral meningitis or brain tumor.

Dull pains are often present in diseases of the spinal cord and sometimes in neuritis. Care should be taken not to mistake them for rheumatism. Sharp, shooting pains are characteristic of neuralgia and often indicate irritation of the posterior nerve roots, as in spinal meningitis and tabes dorsalis. The so-called girdle pain, a feeling as if a band were tied about the body, also indicates nerve-root irritation. The

pain of neuritis may, in some cases, be described as burning; such pain is known as "causalgia." Irritative lesions of the cortical centres may cause pain to be referred to the limbs. Likewise, pain may be referred to remote parts if the nerve supply is the same, as in the knee pain of hip-joint disease, and definite and constant areas of cutaneous tenderness may be due to diseases of the different viscera.[1] When an irritation of one side of the body is felt at a corresponding point on the other it is termed allochiria (p. 48). If the diseased part is superficial, tenderness is often also present, as in neuritis. Pain occurring in an anesthetic area is called analgesia dolorosa.

Paresthesia or Dysthesia (p. 110).—Perverted or abnormal sensations, excepting pain, such as tinglings, crawlings, numbness, itching, etc., are called paresthesiæ. They may be due to organic nerve disease of any portion of the sensory tract from the cortex to the periphery; or they may have a subjective origin. When the touch of one point is felt as two or more it is termed *polyesthesia*. Other forms of perverted sensation have been termed *phrictopathic sensations*[2] (p. 462). *Itching* or *pruritus* is frequently due to toxic irritation of the nerve endings in the skin. *Crawling sensations* or *formication* to a similar cause affecting the nerve trunks (Barker).

SYMPTOMS DUE TO DESTRUCTIVE LESIONS OF THE SENSORY TRACT

These **symptoms** differ according to which pathway is diseased, *i. e.*, that for touch, pain, temperature, or muscle sense. They may be all damaged, or one or more may be and the others not. Sensory fibers often preserve their functions when damaged to an extent that would destroy the functions of the motor fibers, and they recover their function, when lost, sooner than do the motor fibers. Loss

[1] See Head, Brain, 1893, p. 1; 1894, p. 339; 1896, p. 153; and Elsberg and Neuhof, Amer. Jour. Med. Sci., November, 1908, p. 690.

[2] Jones, Jour. Nerv. and Ment. Dis., June, 1908, p. 427.

of sensation may be due to either organic or functional disease.

Anesthesia.—Strictly speaking this word means loss of tactile sensibility, but is often loosely used to include loss of pain sense. When the loss involves one-half of the body it is termed *hemianesthesia*. The face may sometimes be spared. When complete, the entire lateral half of the body, including mucous membranes and special senses, is involved. This is practically always either purely hysterical or hysteria grafted on an organic lesion. Anesthesia of special senses does not occur if the symptom is due to an organic lesion. If of *organic* origin it means the existence of a lesion in the tract between the upper portion of the pons and the cortex of the opposite side (p. 26). A well-defined organic hemianesthesia is due to a lesion either in the posterior part of the posterior limb of the internal capsule or the lateral nucleus of the optic thalamus and lateral homonymous hemianopsia may be associated with it. Crossed hemianesthesia means anesthesia of one side of the body and of the opposite side of the face. It is due to a lesion in the pons. Irregular areas of anesthesia scattered over the body and limbs also occur. They are usually met with in hysteria; sometimes in locomotor ataxia. By *hypesthesia* we mean a diminution, but not complete loss of the power of perceiving sensations of touch.

Analgesia.—Analgesia means loss of sensibility to pain. As has been said, it may occur without loss of tactile sensibility especially in diseases of the cord in which the pathways for touch and pain are widely separated. Loss of pain sense in one lateral half of the body is called hemianalgesia. In some diseases (notably locomotor ataxia) we may have, instead of absolute loss, a delay in the transmission of a sensation, so that it is not felt for an appreciable interval after the reception of the stimulus. There may also be loss of the power of correctly localizing the point stimulated (either touch or pain). Rarely the stimulus is located at an identical corresponding point upon the opposite side of the body. This is termed *allochiria* and is only found in hysteria. When the place stimulated is localized either in

the same or opposite side, but at a widely different point in the body, it is termed *alloesthesia* or *false allochiria*, a phenomenon found in organic disease[1] (p. 48). Other phenomena found in hysteria are *achiria*, in which the patient knows there is a stimulus but cannot tell on which side it is, and *synchiria* when it is referred to both sides. These together with allochiria have been brought under the general term *dyschiria*.[2] Diminution but not complete loss of pain sense is termed *hypalgesia*.

Temperature Sense.—When lost this is usually associated with the loss of sensibility to pain, the two pathways being probably closely related. (See Sensory Tract.) It occurs either as an inability to tell hot from cold; or hot objects may give cold sensations, cold being appreciated, or *vice versa*. Sometimes great differences can be recognized while slighter ones cannot (p. 51). In some affections of the nervous system (notably in syringomyelia, less commonly in diseases affecting the posterior nerve roots, as vertebral caries; cervical pachymeningitis, tabes, spinal tumor, myelitis, multiple sclerosis, and also in disease of the peripheral nerves and hysteria) there may be loss of pain and temperature sense with preservation of the sense of touch, *dissociation of sensation*. When the cord is affected, as in syringomyelia, this is probably due to the fact that fibers conducting pain and temperature sensations cross in the posterior commissure to enter Gowers' tract, and consequently would be destroyed by a lesion destroying the central gray matter, while the fibers conducting tactile sensations pass up the posterior columns and escape. The explanation as to why this occurs in root lesions is not so clear. It may be due to the fibers conducting tactile sensations being less vulnerable to pressure than those conducting pain and temperature sensations.

Muscle Sense.—This includes a recognition of the direction of passive and active movements, of the position of the limbs, and of pressure and resistance.

[1] Jones, Brain, 1907, p. 490, and Camp, Jour. Amer. Med. Assoc., August 22, 1908, p. 666.

[2] Jones, Rev. Neurol. and Psychiat, August, 1909, p. 499, and September, 1909, 559.

Vibrating Sensation.—This is the peculiar vibrating or trembling feeling that occurs when a tuning fork in vibration is placed over subcutaneous bony prominences or surfaces in many parts of the body. The sensation is probably conducted upward in the posterior columns and therefore is present in diseases in which these columns are not affected and lost when they are, often before other forms of sensation are interfered with. It is also lost in disease of the peripheral nerves[1] (p. 53). Its presence or absence may be of service in determining early if the posterior columns or posterior nerve roots are diseased or not.

Stereognostic sense is the name given to memories preserved in the cerebral cortex (parietal lobe) of the characteristics of objects by which we are enabled to recognize them without seeing them. Thus, when a blindfolded person recognizes a dollar placed in the hand he does so by his memory of the peculiar feel, weight, etc., of previous dollars. This sense depends, according to Sailer, upon "an intricate correlation of tactile position, pressure, localization, and temperature perceptions and its interpretation by the higher psychic centres." The most important element seems to be the muscle sense, for it has been found impaired more or less in the reported cases, while other forms of sensibility may be preserved. The stereognostic sense has been found impaired most frequently in hemiplegia. Its impairment is termed astereognosis. It has been proposed by some to limit the term astereognosis to loss of the power of recognizing the shape, size, and form of an object in three dimensions, while loss of the power of telling what the object is, is termed *asymbolia*. The former may be preserved while the latter is lost[2] (p. 53).

SENSORY LOSS—METHODS OF EXAMINATION

Before applying the various tests used to detect sensory paralysis the patient should be blindfolded.

[1] Williamson, Rev. Neurol. and Psychiat., August, 1911, p. 419.
[2] Prince, Jour. Nerv. and Ment. Dis., January, 1908, p. 12.

Anesthesia is tested for by touching the part with a light object, such as a piece of cotton-wool. This may, beginning at the face, be stroked down the body to the feet, telling the patient to advise you as soon as he does not feel it and then marking the spot. In this way the anesthetic areas, if any, may be mapped out. This procedure should be repeated, changing the direction of the stroking to confirm the correctness of the observation. Care must be taken to make as little pressure as possible, as this is a different form of sensibility and may be preserved when tactile sense is lost.[1] In fact, it is probably conducted by other nerve fibers, as the researches of Head, Rivers, and Sherren[2] seem to show. They have probably proved that there are three systems of afferent fibers in the peripheral nerves:

1. "Those which subserve deep sensibility and conduct the impulses produced by pressure and movement of limbs. The fibers of this system run mainly with the motor nerves and are *not* destroyed by division of all the sensory nerves of the skin." These fibers also run in the tendons. If the pressure is severe enough pain may be produced.

2. Those which subserve *protopathic sensibility, i. e.*, painful stimuli and extremes of heat and cold, but inability to localize the spot stimulated.

3. Those which subserve *epicritic sensibility,* by which we gain the power of cutaneous localization, of the discrimination of two points, and of the finer degrees of temperature, as cool and warm.

Protopathic sensibility returns first in the case of an injured sensory nerve. These observations should be remembered especially in testing loss of sensation after injuries to the peripheral nerves.

When the lesion is in the spinal cord the phenomena are different,[3] as follows:

[1] For very accurate measurement the v. Frey hair esthesiometer may be used. The hairs are graduated to exercise different known pressures, and hence accurate comparisons can be made. Trotter and Davies (Rev. Neurol. and Psychiat., 1907, p. 761, and Brain, 1911, p. 92) recommend that the patient outline the area with his eyes open and using his own finger to make the touches. This could only be done in organic disease.

[2] Brain, 1905.

[3] See Head and Thompson, Brain, 1906, pp. 537-741.

1. If sensibility to pain is abolished *all* forms of painful stimuli are simultaneously affected.

2. Sensibility to heat may be abolished with coincident disturbance of that to cold, and *vice versa*. When sensibility to heat is disturbed in consequence of an intramedullary lesion the patient no longer appreciates any thermal stimulus between 30° and 60° C. That is to say, insensibility is absolute for both intermediate and extreme degrees. Insensibility to heat and cold may be absolute, and yet the patient may be able to recognize the lightest tactile stimulation and to discriminate the two points of the compasses.

3. If sensibility to touch is abolished in consequence of an intramedullary lesion all forms of tactile stimuli will be found to be affected. The peripheral afferent impulses for touch and pressure arriving by way of the epicritic and deep systems become combined in the spinal cord.

4. In intramedullary lesions every other form of sensibility may be abolished in a part which still remains sensitive to passive movement. Or the patient may be unable to recognize even the greatest passive movements, although sensitive to all tactile and passive stimuli; or the sense of passive position and movement may be disturbed in one leg, while every other loss of sensation may be found in the leg of the opposite side.

5. Within the spinal cord the impulses which underlie the power of discriminating two points are separated from those of tactile sensibility.

Analgesia is discovered by pricking the skin and mucous membrane with a pin, going over the body systematically, as in the test for anesthesia, and instructing the patient to inform you as soon as he feels it, or else to count each time he feels the prick. If he misses a count we know that the patient does not feel it. It should also be noticed if the patient winces, or if there is delayed transmission, indicated by the announcing of the sensation being felt several seconds after the stimulus is used. It should also be determined if he is able to localize the place touched or pricked and the distances apart at which two points can be recognized (p. 51). If it is desired to make accurate comparisons some form of

algesimeter may be used. In those the amount of pressure on the point necessary to evoke a response, if any, can be determined.

Loss of temperature sense is discovered by the alternate use of test-tubes filled with hot and cold water.

If **loss of muscle sense** is suspected we test the perception of passive movements by changing the position of the segment of a limb and instructing the patient to indicate when he can feel the movement. It is well to test first the fingers and toes. The patient should also be asked to indicate the direction of the movement, if its occurrence is recognized. Normally both the movement and its direction are recognized simultaneously. In some conditions, however, as cerebral lesions, a larger range of movement may be necessary for its direction to be appreciated than for its occurrence.

Perception of pressure and *resistance* may be tested by using rubber balls of the same size filled with different quantities of shot. These are placed on the hand or leg, and the patient is asked to determine which is the heavier. A difference of $\frac{1}{40}$ of the total weight should be detected if normal. Pressure sense may also be tested by making pressure on or pinching the part with the hand. Head has discovered that in disease of peripheral nerves, pain produced by excessive pressure may be experienced when the prick of a pin is not felt (p. 51).

Knowledge of the *position of parts* is determined by placing a limb in a certain position and asking the patient either to place the opposite one in the same position or to describe it. Another method is to ask him to touch with the normal hand some definite spot, as the great toe, end of finger, etc. The *presence* or *absence* of the *stereognostic sense* is determined by placing familiar objects in the hand and requesting the patient to name them; if he cannot there is loss of the sense. It is also important to ascertain if he can describe the shape, consistency, and roughness or smoothness of an object (p. 50).

Vibrating sense (p. 50) is tested by placing a large tuning-fork over bony prominences, as the malleoli, inner surface of the tibia, the sternum, palms of the hands and soles of the

feet, styloid process of the ulna, nails of the fingers and big toes, and anterior superior iliac spines. In making the first test the patient should be asked to describe what he feels. If he answers a vibrating or trembling sensation we know that he recognizes the nature of the stimulus.

INCOÖRDINATION—ATAXIA

Definition.—For a muscular movement to be performed in a regular, smooth, and coördinate manner it is necessary that the contraction of each muscle concerned in the movement, and also that of the antagonists of these muscles, be accurately *proportioned* in force and time. For example, when we use the *flexor* muscles or a limb the *extensors* act as a *balance;* otherwise all movements would be quick and jerky (p. 43).

For the proper performance of this function, sensations travel from the muscles, articular surfaces, and tendons (muscle sense), and to some extent from the skin (tactile sense) up through the posterior columns and direct cerebellar tract to the cerebellum and thence to the sensori-motor region of the cortex (Figs. 9 and 10). A lesion so situated as to prevent these sensations reaching the cortex will produce *incoördination.* Hence lesions of the brain, cord, or peripheral nerves may produce this symptom, which is usually known as *ataxia.* More properly the use of the term *ataxia* is only applied to that form of incoördination due to spinal-cord disease.

Ataxia may also be present during apparent rest, and is then called *static ataxia.* To maintain any posture, coördinated muscular movements are necessary. When this function is deranged, swaying movements of the body or irregular movements of the limbs become manifest.

Incoördination.—Cerebral.—Cortical lesions, especially of the parietal lobes, may produce more or less disturbance of the muscle sense and consequent ataxia. This is often associated with hemiplegia, and is then known as posthemiplegic ataxia. The incoördination which sometimes occurs in *hysteria* is also of cortical origin.

Cerebellar.—Lesions of the cerebellum, especially of the middle lobe, cause a form of incoördination commonly termed *titubation*. The gait resembles that of alcoholic intoxication; the patient is unable to walk in a straight line, and there is often a tendency to fall always in the same direction, either right, left, or backward, as the case may be. The arms are not affected so commonly as the legs. Related to this form of incoördination are the symptoms known as asynergia and diadococinesia (p. 356). Similar disturbances sometimes occur in disease of the pons or medulla in which either the *fillet* or cerebellar peduncles are involved.

Spinal Cord and Peripheral Nerves.—In the great majority of cases the symptom is due to disease, either of the posterior columns, posterior nerve roots, or both. The patient walks in such cases, if the symptom is marked, with his eyes fixed on the gound; the legs wide apart; lifts the feet higher than normal and throws them forward in a jerky manner (Fig. 56). In some cases of multiple peripheral neuritis, if the paralysis is not marked, incoördination is present.

Examination.—The existence of *static ataxia* is discovered by causing the patient to hold the limbs in some fixed position without support, when the movements described on page 54 will be seen; or to stand erect with the feet close together and the eyes closed. If the symptom is present the patient will sway violently to and fro and may fall. This is known as the *Romberg sign.* It is also spoken of as the station. Thus, if he stands properly his station is normal; if he sways the station is said to be poor.

Dynamic ataxia of the lower limbs is tested by making the patient walk forward and backward with the eyes first open and then closed; turn suddenly while walking; raise himself upon his toes with the eyes closed; and stand on one leg. When in the recumbent position have him endeavor to place the heel of one foot upon the opposite knee with the eyes closed. If incoördination is present this is done awkwardly or not at all (heel to knee test).

The *upper limbs* are studied by having the patient touch the end of his nose with his finger with the eyes closed (finger

to nose test); pick up small objects; button his clothes; and after separation of his arms, making him endeavor to bring the points of his fingers together. Inability to perform these movements will indicate ataxia. Degrees of incoördination so slight as not to be noticeable in the gait are detected by examining for the Romberg symptom with the feet and legs of the patient bare; in the effort to maintain his position contractions of the muscles of the legs will be caused, which can be detected by watching the tendons about the ankles.

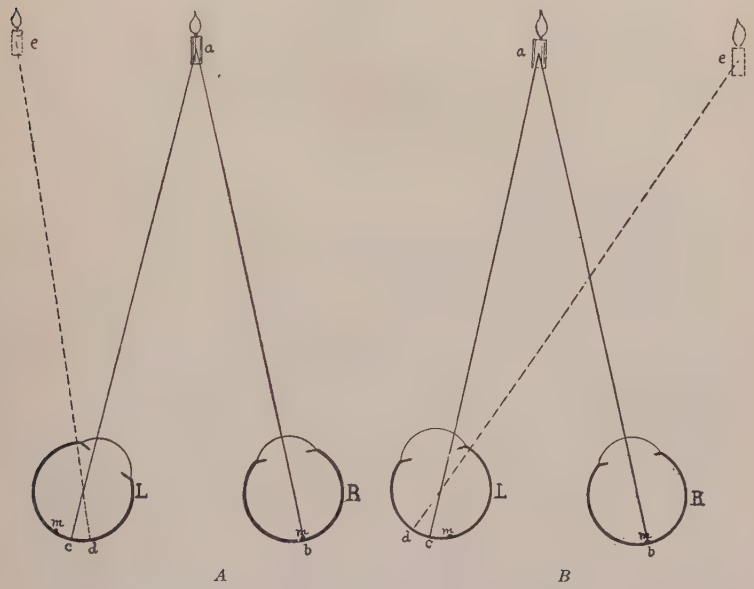
EXAMINATION OF SPECIAL SENSES

Sight.—In diseases of the nervous system the sense of sight may be variously affected, the causes of which are either functional or organic. When there is a subnormal acuteness of vision we term the condition *amblyopia* (p. 137). Diminution of vision in one eye due to lesion of the optic tract of the opposite side has been termed crossed amblyopia; in these cases there is also a much slighter diminution of vision in the eye of the same side (p. 35).

Seeing two objects when looking at one (double vision) is known as *diplopia*. If persistent for any length of time this symptom indicates weakness of one or more of the ocular muscles (pp. 144, 146, 148). Delicate tests employed by the ophthalmologist may be required to ascertain which. It can be usually determined by asking the patient to fix the moving finger of the examiner with both eyes. As soon as two images appear it should be at once stated. A lighted candle may be used instead of the finger, and one eye may be covered with colored glass to further distinguish the images. The image seen by the sound eye is called the true, that seen by the affected eye the false image. If the latter is on the same side as the eye by which it is seen, *i. e.*, if the right image disappears with closure of the right eye, it is called *homonymous diplopia*, otherwise it is spoken of as *crossed*. Usually convergent strabismus is accompanied by homonymous, and divergent strabismus by crossed diplopia (Fig. 16). General rules are: When in moving the eyes in a given direction the

double images begin to separate, that image which moves the faster and the farthest belongs to the paralyzed eye. The image belonging to the paralyzed eye is displaced in just that direction in which the affected muscle if intact would

FIG. 16



A, convergent squint. The ray of light, *a c*, falls to the right of the sensitive spot, *m*, of the inturned left eye, *L*. Such stimulus has always been recognized by the mind as emanating from objects to the left, and not realizing the disordered position of the ocular globe a false image, *e*, is projected an additional distance to the left, measuring from *c* as the operating sensitive spot, in the line *d e*. *B*, divergent squint. The ray of light, *a c*, falls to the left of the macula, *m*, of the outwardly turned left eye, *L*. Such stimulus has always been associated with objects to the right, and the false image, *e*, is projected to the right, *c*, acting on the sensitive spot at the time. (Church and Peterson.)

naturally move the eye.[1] A monocular diplopia, *i. e.*, seeing two images with one eye, has been described in hysteria (p. 472).

[1] Eye and Nervous System, Posey and Spiller, p. 215.

Amaurosis is a word sometimes used to express blindness. *Hemianopsia* denotes blindness of one-half the visual field. *Homonymous hemianopsia* indicates blindness of the outer half of one and the inner half of the other field of vision. When we wish to indicate which side of the retina is blind we use the word hemiopia: thus, a right-sided hemianopsia would be a left-sided hemiopia (Fig. 34).

In all doubtful cases the eye should be examined by a skilled ophthalmologist, the presence of either optic neuritis or atrophy, primary and secondary, being valuable symptoms of many organic diseases of the nervous system. The existence of hemianopsia can be roughly determined by sitting the patient in front of you with one eye blindfolded, and telling to him look steadily at the end of your nose with the other. A white object is then brought from different points of the periphery toward your nose, and the patient is instructed to say when he sees it. If he does not, that part of the field is blind. When patients are stupid, either from disease or naturally, the following test may prove useful, *i. e.*, an article of food or something in which the patient would naturally take an interest is brought into what should be the patient's field of vision; if it is seen he will instinctively turn the eyes toward it, but if blindness is present no attention will be paid to it.

The existence of *refractive errors*, a common cause of headache, may be surmised if the eyes water and letters run together after reading a while. The presence or absence of exophthalmos should also be noted.

Hearing.—This, to be accurately tested, also requires the services of a specialist. The existence of tinnitus should be inquired for. The acuity of hearing can be determined by blindfolding the patient and covering one ear. A watch is then held some distance from the other ear and made to approach toward it, the patient signifying when he hears the tick. Watches, of course, differ in the loudness of their tick; the normal distance being from two to four feet. In order to determine whether the deafness, if any, is due to disease of the nerve or of the middle ear, we use a large tuning-fork (note C), and determine whether the sound is

heard best when it is held at a distance from the ear or when placed on the top of the head. If aërial conduction is the better, the nerve or its endings are diseased; if the sound by bone conduction is more distinct, the cause of the deafness exists in the middle ear (Rinne's and Weber's tests).

Smell.—Care should be taken in testing smell not to use odors which are irritative, as ammonia. With one nostril closed we test the other with peppermint, asafetida, or some non-irritating perfume. The possibility of catarrhal trouble causing loss of smell must always be considered.

Taste.—In testing taste we use a bitter substance, as quinine; a sweet one, as sugar; and a sour one, as vinegar. The bitter should be used last. The patient keeps the tongue protruded, while one of the solutions is painted with a camel's-hair brush, on each side of the tongue, first posteriorly and then anteriorly. The patient can indicate if he tastes the solution by pointing to the name, which has previously been written on paper. Under no circumstances should the tongue be withdrawn into the mouth until the test is completed. To decide if there is complete loss of the sense it should also be tested on the lips, the inner surfaces of the mouth, the palate, the pharynx, the epiglottis, and, when possible, the larynx.

THE REFLEXES

To **cause a reflex action** there are necessary an *afferent nerve,* either an ordinary sensory or the special excitoreflex nerve supplied to the viscera; an *efferent* or *motor nerve;* and the cells (reflex centre) in the gray matter of the cord or medulla to which the afferent nerve runs and from which the efferent starts. This *mechanism* is controlled by the higher cortical (motor) cells, which exercise their influence through the pyramidal tract (Fig. 17). The indirect motor tract probably also takes part in controlling the deep reflexes (p. 25). It does this through its action in maintaining muscle tone (p. 36). Loss of this influence causing hypotonicity or relaxation of the muscles and either loss or diminution of these reflexes.

Reflexes are of *three kinds:* the skin, or superficial; tendinous and muscular, or deep; and visceral.

When any of the components of the reflex arc—viz., afferent nerve, cells in gray matter, efferent nerve—are damaged, the reflexes under the control of that particular arc are lessened or absent. An irritative lesion of the pyramidal tract will sometimes, by increasing inhibition, cause a reflex to be diminished or absent. If the inhibitory influence of the brain is removed, as by a destructive lesion of the

FIG. 17

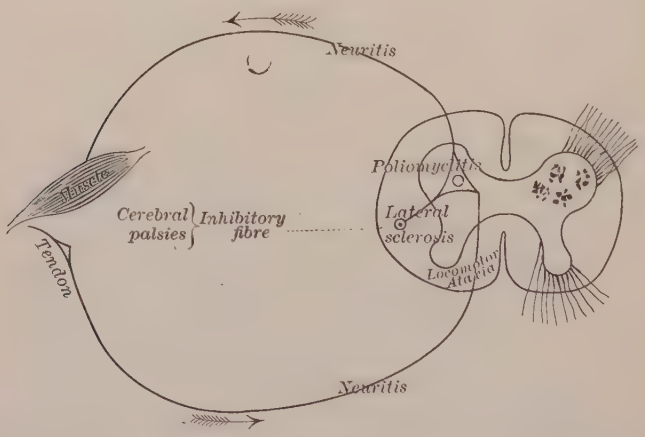


Diagram showing reflex arc and inhibitory fiber, and some of the diseases which exaggerate or destroy the knee-jerk.

pyramidal tract, the reflexes are increased. Also irritation of a peripheral nerve may sometimes cause an increased reflex in the area supplied by it, thus increased knee-jerks may be present in the early stages of multiple neuritis even when the nerves of the legs are affected. Later, in such cases, they are absent.

The Skin Reflexes.—These are **produced** by scratching, irritating, or tickling the skin, which, if the reflex is present, causes a contraction of the muscles near the irritated part.

With the exception of the plantar and cremasteric, these reflexes are not always present in normal individuals.

The most important are:

The **plantar reflex** consists of a flexion of the toes upon the metatarsus and a quick, involuntary jerking of the foot and leg when the sole of the foot is irritated. In making this test the leg should be in such a position as to insure muscular relaxation, the foot should be warm and the sole of the foot stroked with a moderately sharp instrument, as a toothpick, from the heel to the toes, preferably on the outer side. M. J. Babinski has shown that if a lesion of the pyramidal tract which involves those fibers which go to the leg[1] is present, instead of flexion of the toes, there is a slow extension of either all the toes or only the great toe. This has been termed the Babinski reflex or phenomenon. It has also been seen in cases in which there is irritation of the motor tracts without destruction of nerve fibers, as in typhoid fever,[2] tetanus, uremia, etc. In children under one year of age the extensor response is normal.

A similar action of the toes in similar pathological conditions is seen in *Oppenheim's, Chaddock's,* and *Gordon's reflexes.* The first is elicited by stroking down the inside of the leg just back of the tibia with a blunt instrument, the second, by irritating the outer side of the foot below the malleolus, the third, by grasping the relaxed muscles of the calf with the hand and squeezing them.

The **cremasteric reflex** is caused by irritating the skin on the inside of the thigh, when a drawing up of the testicle will be seen.

The **abdominal reflex** consists of a contraction of the rectus muscle of the abdomen when the side of the abdomen is scratched.

The **lid reflex** consists of closure of the eye when either the conjunctiva, cornea, or retina is irritated.

The **pupillary skin reflex** consists of a dilatation of the pupil when the skin on the side of the neck is pinched.

[1] Potts and Weisenburg, Rev. Neurol. and Psychiat., 1910, 576.

[2] Levi, Revue Neurologique, November 15, 1900, p. 1005.

The **supra-orbital reflex** of McCarthy is elicited by striking with a percussion hammer the supra-orbital foramen when a contraction of the lower half of the orbicularis palpebrarum will be seen. Its absence indicates destructive lesion of either the supra-orbital or facial nerves.

Those which are of not so constant occurrence are:

The **epigastric reflex,** produced by irritating the skin of the lower part and side of the thorax. This causes contraction of the upper fibers of the rectus muscle.

The **erector spina reflex** consists of contraction of some of the fibers of the erector spinæ, caused by irritation of the skin along its outer edge.

The **scapular reflex** consists of a contraction of the scapular muscles when the skin over them is irritated.

The **palmar reflex** is produced by irritating the palms of the hand, when a jerking away of the arm and closing the hand will be seen.

The Deep or Muscle and Tendon Reflexes.—Some of these are not found in healthy individuals, only being present when reflex activity is increased. Only the more important of them are mentioned here. It should be remembered that their absence may sometimes be caused by mechanical reasons, as ankylosis of joints or when there is excessive spasticity of the muscles, they being so contracted that further contraction is impossible.

The **knee-jerk** or patellar tendon reflex is the most constant, but rarely it is not present in apparently healthy individuals. It consists of a sudden contraction of the quadriceps femoris and vastus internus and externus muscles, produced by striking the patellar tendon while the leg hangs loosely at a right angle to the thigh; for instance, the leg being crossed over the other, or the patient sits so that the legs hang over the edge of a table. Complete relaxation is most important. It may be secured by making the patient look at the ceiling while being seated. This reflex may also be produced if the lower part of the muscle is struck. It may be increased, or at times brought into activity when absent—called reinforcement—if at the time the tendon is struck the patient performs some voluntary

muscular movement, such as tightly closing the hands or pulling on his clasped fingers at the instant the tendon is struck (Jendrassik's method). Reinforcement is not possible if the absence of the reflex is due to organic disease. When the reflex is greatly increased a *patellar clonus* may be caused. This consists of rapid clonic contractions of the quadriceps muscle when the patella is either suddenly pushed downward, or being held in this position the finger holding it is percussed.

The **tendo Achillis** or **ankle-jerk** is elicited by striking the tendo Achillis, which causes contraction of the soleus muscle and a plantar flexion of the foot. It is best elicited by causing the patient to get upon his knees on a chair or bench so that the feet just project over the edge, and then striking the tendon just above its insertion. Another method is for him to stand upon one leg, the one tested being flexed on the thigh and supported by the examiner. If the patient is bedfast it may be tested by elevating and supporting the leg slightly from the bed, then pressing the foot slightly backward and striking the tendon.

Ankle clonus is caused by grasping the slightly flexed leg under the calf with one hand and suddenly making dorsal flexion of the foot with the other and holding it in that position. When it is present there will be seen a series of rapid contractions of the soleus muscle causing to-and-fro movements of the foot. Its presence means a lesion of the pyramidal tract, and is never present in health and is rarely present in functional disease. In these a few irregular oscillations may sometimes occur which have been termed *pseudo-ankle clonus*.

The **wrist-jerk** is elicited by striking the flexor tendons at the wrist, made prominent by passive extension of the hand, the wrist of the patient being supported by the examiner. Flexion of the hand takes place.

The **elbow-** or **triceps-jerk** consists in a contraction of the triceps muscle when its tendon is struck just above the elbow-joint, the forearm hanging loosely at a right angle to the supported arm.

The **biceps-** and **supinator-jerk** is obtained by striking the lower end of the radius, the elbow being partly flexed at the

time or by slightly flexing and supporting at a right angle the forearm of the patient on the arm of the examiner, who places his thumb over the biceps tendon making light pressure upon it. Percussion is then made upon the dorsum of the thumb. Contraction of the biceps results.

The **jaw-jerk** is produced by causing the patient to sit with the mouth partly open with a ruler or something similar laid across the lower teeth. When this is struck the masseter muscles contract and the mouth closes. The jaw-jerk always indicates disease.

A **muscle-jerk** is obtained by striking the stretched muscle, when certain or all of the fibers contract. It may be present or increased in diseases in which the tendon reflexes are absent; for instance, in progressive spinal muscular atrophy.

The Visceral Reflexes.—The Eye.—Important reflexes are discovered by examining the eyes. The *light* reflex is produced by throwing a bright light into the eye, when the pupil contracts, to dilate again when the light is removed. When the light enters the observed eye it is termed the *direct light reflex;* if it is thrown into one eye and the observed eye is shaded, it is known as the *indirect* or *consensual light reflex.* The reflex arc consits of fibers running from the retina to the third nerve nucleus (anterior portion), and from there to the iris. To roughly determine the presence of the direct light reflex, the eye not being tested is covered, the patient being in a dark room, while the other eye is exposed to a bright light, or the tested eye may be covered for a brief period and then uncovered, the patient being in a lighted room. The consensual light reflex may be tested by placing the patient facing a window, one eye being screened (the one tested) in such a manner that it will be in shadow, but so that the examiner can note the movements of the iris. The exposed eye is then alternately covered and uncovered, and if the reflex is present the iris of the screened eye should contract and dilate equally with that of the exposed one.

Sometimes after the pupil contracts it will dilate again, although exposed to the same light, and then contract, doing this alternately for several times. This phenomenon

is termed *hippus*. It may occur in neurasthenia, hysteria, epilepsy, and organic diseases of the brain—a mild degree of it is normal. Lesions of the brain posterior to the reflex centres (p. 135) do not cause loss of the light reflex (Fig. 34). If, however, the lesion is in the optic tract of one side anterior to or in these centres there is loss of the pupillary light reflex when light is thrown on to the blind half of the retina only (Wernicke's hemianopic pupillary reflex or inaction sign). The test requires much skill to make properly. When the eye looks at a far object the pupil normally dilates, and contracts when looking at a near one; this is known as the convergent and sometimes as the accommodation pupillary reflex. Each eye should be tested separately, the other one being covered. In some cases of organic disease of the nervous system the pupillary light reflex is lost while that for convergence is preserved; this is known as the *Argyll-Robertson pupil*, and is a frequent symptom of tabes dorsalis and paresis; occasionally it may occur in other diseases affecting the brain. This phenomenon may also rarely be reversed, the light reflex being present and that for convergence lost. In old people the convergence reflex is frequently diminished normally. These phenomena may be unilateral.

The Reflexes of the Bladder, Rectum, and Sexual Apparatus.—Urination and defecation are reflex acts under the control of the higher centres.[1] If the inhibitory influence is removed, and voluntary control over the sphincters is lost, urine and feces are expelled as soon as the bladder or rectum is full. This condition is seen to greater or less degree whenever the function of the pyramidal tracts is interfered with. If this condition persists, weakness of the detrusor muscles occurs after a time and the bladder is not completely emptied. If, in addition to the preceding conditions, the tone of the *compressor urethræ* muscle is intact, there follows retention of urine until the bladder distention is sufficient to overcome this tone. Then we have the *incontinence of overflow*.

[1] It is believed by some, notably Müller, that the centres for urination and defecation are not in the sacral region of the cord (p. 68), but are in the pelvic sympathetic ganglia. The cells in the cord are merely way stations in the neurone system.

If any of the components of the reflex are involved, incontinence also takes place (Fig. 18).

If the *motor part* is diseased, the *sphincters* are relaxed.

In the case of the *rectum* this condition can be differentiated from the incontinence due to loss of inhibition by introducing the finger into the rectum. In the former case the sphincter will be found relaxed; in the latter it grasps the finger firmly (anal reflex).

FIG. 18

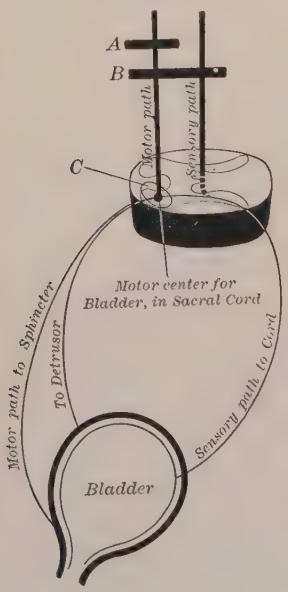


Diagram illustrating the innervation of the bladder and the effect of lesions in various parts of the spinal cord upon the function of micturition. A lesion, *A*, which interrupts the voluntary path to the bladder centre in the sacral cord causes incontinence of urine. When a sufficient quantity of urine accumulates in the bladder there occur a reflex contraction of the detrusor and a relaxation of the sphincter. The sensory path from the cord to the brain being uninvolved the patient is conscious of the process, but cannot exercise voluntary control over it. With a lesion, *B*, which involves also the sensory path, the patient is unconscious of the filling and reflex emptying of the bladder. A lesion, *C*, which causes destruction of the sacral reflex centre of the bladder causes continuous dribbling of urine, and not its automatic expulsion at intervals. (Herter.)

When complete paralysis is not present there is difficulty in starting the stream of urine, or there may be obstinate constipation.

When the *sensory part* of the arc is damaged the patient is unaware that the bladder or rectum is full (Fig. 18).

Damage to the reflex arc governing the *genital functions* causes loss of the power of erection of the penis and loss of desire. Removal of the inhibitory influence may produce

priapism. The reflex centres of these functions are in close proximity. The location in the cord of all of the various reflex centres and their localizing significance will be found under Localization in the Spinal Cord (p. 368).

SYMPTOMS DUE TO TROPHIC, VASOMOTOR, AND SECRETORY DISTURBANCES

Trophic disturbances consist of atrophy of muscles; of cutaneous, osseous, and mucous tissues; fragility of the bones; joint affections, known as *arthropathies* or Charcot joints. The latter are characterized by effusion into the joint and separation of the articular surfaces; in extreme cases thickening of the ends of the bones ensues, to be followed later by atrophy, disappearance of the cartilage and erosion of the ends of the bones and undue mobility of the joint. Pain and redness are, however, absent. Other trophic symptoms are the perforating ulcer of tabes, various skin eruptions, as herpes; pigmentation; alopecia, and bed-sores.

The nerve cells of the anterior horns are the trophic centres for the muscles. The posterior nerve roots and posterior horns of the cord have to do with the nutrition of bones, joints, and skin. The cells of the posterior root ganglia also have an influence over the skin (p. 232).

Vasomotor symptoms are pallor and coolness of the skin, due to spasm; and edema and cyanosis, due to relaxation of the bloodvessels. The latter may also be due to spasm (p. 494). There may be an unsteadiness of action of the vasomotors, they dilating and contracting in an irregular way, thus causing various flushings and localized edemas (angioneurotic edema), Raynaud's disease, erythromelalgia, etc.

Secretory disturbances consist of increase or diminution of glandular secretions, as excessive sweating or hyperidrosis, excessive dryness or anidrosis. There may be peculiar odors or colors of the secretion. These usually indicate derangement of the sympathetic system. Either *irritation* or *paralysis of the cervical sympathetic* cause characteristic symptoms often of service in the diagnosis of lesions of the cervical

region of the cord or of the nerve roots originating there. The spinal origin of this nerve is probably in the lower cervical and first dorsal segments of the cord. A destructive lesion causes retraction of the eyeball, narrowing of the palpebral fissure, absence of dilatation of the pupil when shaded or when cocaine is instilled, some pallor of the face and neck, dryness of the nostril and mouth on the affected side, and absence of or a diminished secretion of sweat in the same region, and in addition on the arm and chest. Irritation of the nerve causes just the opposite of the above symptoms, *i. e.*, protrusion of the eye, dilatation of the pupil, flushing, and increased secretion.

The sympathetic system is closely related with the spinal cord and nerves. The cells of the *intermedio-lateral tract*, probably are connected with it.[1] Vasomotor and secretory centres are believed to be situated here. The anterior nerve roots contain vasoconstrictor nerves, which pass from these centres to the sympathetic nerves. Vasodilator nerves also leave the cord to join the sympathetic. Fibers pass to the sweat glands in the same way. By means of this tract and its connections lesions of the cord may exert an influence upon visceral functions (p. 364).

SCHEME FOR THE EXAMINATION OF PATIENTS

A thorough examination is of the utmost importance in all cases of nervous disease, not only of the nervous system but of all the organs of the body, especially the cardiovascular, pulmonary, urinary, and digestive. It is always useful to first record the chief complaints of the patient, next get the *family history*, especially as regards the evidence of nervous and mental disease, the nature of it if present; the existence of gout, rheumatism, tuberculosis, cancer, alcoholism, syphilis, and consanguinity of the parents.

Social History.—Previous occupations, habits as regards alcohol, tobacco, tea, coffee, sexual excess, overwork, mental strain, or shock. Existence of previous nervous disease,

[1] Bruce and Pirie, Rev. Neurol. and Psychiat., 1907, p. 1.

especially fits, chorea, headache; syphilis, gout, or rheumatism. If a woman, the number of children, health of living ones and cause of death of those dead, number and cause of miscarriages.

Examination.—General appearance, description, and location of deformities, scars, etc. Gait and station, if able to walk and stand.

Mental Functions.—Memory, attention, incoherence, orientation, emotions, hallucinations, delusions, delirium, coma, drowsiness, insomnia, irritability.

Abnormal Movements.—Convulsions, if present, describe the character of and note the existence of aura, biting of the tongue, unconsciousness, drowsiness, or delirium. If nocturnal or not, involuntary micturition. Tremor, note if rapid, slow, coarse, or fine, location of, if it ceases after voluntary effort or is increased by it. Choreiform movements if present, location of, mode of onset, ceasing or not during sleeep. Local spasmodic movements or tics, muscles affected, character of movement, locomotor effect or not, presence of pain. Athetoid movements.

Speech.—Character of, ability to pronounce test sentences. (See p. 416.) If due to cerebral lesion (aphasia). (See p. 306.)

Cranial Nerves.—*Olfactory*—anosmia, parosmia. *Optic* nerve and *motor* nerves of eyeballs, vision, fields, hemianopsia, condition of the optic disks, size and equality of the pupils, their reaction to light (direct and consensual), and convergence, hippus, movements of the eyeball, separately and in association. Inequality in size of palpebral fissures, ptosis, diplopia, nystagmus, asthenopia.

Fifth Nerve (Motor).—Muscles of mastication. Fifth nerve (sensory). See Sensory Disturbances, pp. 47 to 49.

Seventh Nerve.—Muscles of face. Note whether complete or only lower part affected and if there is loss of taste on the affected side. Electrical examination.

Eighth Nerve.—See Sensory Disturbances, p. 58.

Ninth Nerve.—Ability to swallow. If impaired, note if liquids or solids can be swallowed best.

Tenth Nerve.—Movements of vocal cords, if speech is affected or breathing interfered with.

Eleventh Nerve.—Action of sternomastoid and trapezius muscles.

Twelfth Nerve.—Ability to protrude the tongue, direction in which it is protruded, ability to move it from side to side, atrophy, tremor.

Spinal Nerves (Motor).—Test all muscles and movements; note if completely lost or only weakened, character of respiratory movements. If muscles are spastic, or flaccid, existence of hypotonia, fibrillary tremors, atrophy (if this is present, electrical examination).

Reflexes.—Conjunctival, ciliospinal, palatal, epigastric, abdominal, cremasteric, plantar, existence of the Babinski phenomenon. Jaw, biceps, triceps, wrist, knee, Achilles jerks (increased, decreased, absent, or normal), patellar clonus, ankle clonus.

Incontinence of urine (paroxysmal or dribbling), retention, difficulty in passing (examine prostate of old men), constipation, difficulty in controlling feces (if present, test the anal reflex), consciousness of desire, sexual power, desire, priapism.

Sensory Symptoms.—Pain, location of, character of, tenderness (to deep or light pressure), location of, paresthesia. Vertigo (subjective or objective), direction of movements.

Absence or *diminution* of touch, pain, vibrating and temperature senses, localization of place touched, testicular sensibility. *Muscle sense*, ability to tell direction of passive movements of limbs, fingers, or toes, ability to recognize position of limbs, heel to knee, finger to nose tests. Differences in pressure and estimating differences in weights, recognizing existence of muscular contractions caused by electrical current. Astereognosis, apraxia (p. 309).

Taste, especially if complete facial palsy or anesthesia of the face is present. On what parts of tongue loss is present.

Hearing, if deaf, whether due to nerve or middle-ear disease.

Trophic Symptoms.—Herpes, bed-sores, enlarged joints, perforating ulcers, glossy skin, spontaneous fractures, pes cavus, etc.

Sympathetic nerves, absence of dilatation of pupil when shaded, narrowing of palpebral fissure, exophthalmos, pseudoptosis, unilateral sweating or undue dryness of secretions, flushings, comparative pallor.

If evidences of meningitis or subdural hemorrhage are present a lumbar puncture and Wassermann test should be made (p. 94).

While it may not be necessary to investigate all of these symptoms, in every case it is a safe rule to do so, noting not only those which are present, but also if they are not. Further help in studying cases may be had by referring to Chapters V, VI, VII, VIII, and Cerebral and Spinal Localization.

CHAPTER IV

GENERAL THERAPEUTIC MEASURES

ELECTRICITY

Physics.—Potential.—The laws which govern the electrical current are very similar to those which govern the flow of water. If we have two vessels of water one above the other and connected by a pipe, the fluid in the higher one will endeavor to get to the lower; the water in so doing will exert force and consequently a *capacity to do work*, which is called its *potential*. The fluid in the lower vessel would also endeavor to reach a lower level which is ultimately the level of the sea, and in so doing would exert force, but not so much as that exerted by the higher body of water. The sea level would be zero potential. The difference in the force exerted by these two bodies of water would be their difference in potential.

By means of certain agencies—viz., friction, chemical action, etc.—electricity is separated into *positive* and *negative* electricity, the positive being of higher potential than the negative; these tend to become united, that of the higher potential tending to flow toward the lower, just as the fluid in the higher vessel tends to flow toward the lower; and this tendency, when the conditions are favorable, causes an electrical current. The zero point of electrical potential is the earth.

The Volt.—The force which starts the column of water flowing is that of *gravity;* that which causes the electrical current to flow is termed the *electromotive force* (*E. M. F.*) and the *unit of measurement of such force* is the *volt.*

The Ohm.—Certain substances are much more pervious to the passage of water than others; so with the electrical current; substances through which the current will flow

with facility are known as *conductors*. In the order of their relative value they are the metals, charcoal, plumbago, dilute acids, saline solutions, water, living animals, and flame. Those which do not permit its free passage are called *insulators*, or *non-conductors*. They are rubber, silk, glass, wax, sulphur, resins, shellac, and dry air. As the water flows through the pipe it meets with resistance; so does the electrical current, as it flows through the conductor, and this resistance, similar to that which water would experience, will depend on the length, composition, and area of cross-section of the conductor, *i. e.*, a current passing a short distance through a good conductor with a large area of cross-section will meet with less resistance than will a current subject to opposite conditions. The *unit of resistance* is the *ohm*.

The Ampère.—The actual *current strength* (C) depends on *Ohm's law*, viz., the current strength is equal to the E. M. F. divided by the resistance $\left(C = \frac{E.M.F.}{R.}\right)$. Current strength is measured in *ampères;* and in medicine we use the one-thousandth part of an ampère or a *milliampère*.

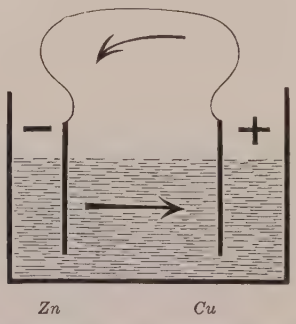
As water may be *confined*, so by means of insulators can electricity. It is then said to be *static*.

The **electric currents** used in medicine are of five kinds, viz., *static, galvanic, faradic, sinusoidal*, and *high frequency*. The **static machine**, which generates electricity by friction, develops a very high electromotive force, and consequently the electricity is of high potential. When the patient is insulated from the ground and connected with one of the conductors he becomes enveloped in a layer of electricity which is confined by the dry air. When the other electrode is approached the current flies to the zero point, just as a large volume of water, which had been dammed, would do when the dam bursts. It does so with such force that it leaps through space and, by igniting the small particles from the conductors, causes a *spark*.

The **galvanic** or **constant current** is commonly produced by chemical action upon two dissimilar substances. The current flows from the substance most acted upon (the positive

element) to that least so (the negative element); and if they are connected by a conductor, from this back again to the positive. Such a combination of substances is termed a *cell* (Fig. 19). A combination of *cells* constitutes a *battery*. The point from which the current leaves the cell (the negative element) is called the *positive pole* or anode; and that where it enters the positive element, the *negative pole* or cathode. This constitutes a *circuit*. So long as the circuit is closed a constant E. M. F. is maintained and the current will flow; when the circuit is open the current ceases.

Fig. 19



This represents a vessel filled with dilute H_2SO_4, into which plates of zinc and copper are immersed and connected by a piece of wire. The current starts at *Zn*, flows to *Cu*, and hence through the wire to *Zn* again. *Cu* is the positive pole or anode; *Zn* the negative pole or cathode.

The **faradic current** is produced by *induction, i. e.*, if we make a coil out of the wire which connects the two elements of a cell and surround this coil with another one having no connection with it, and open and close the circuit in the first or primary coil, an E. M. F. will be generated which will cause a current to flow in the second or secondary coil. This current is but momentary, only occurring when the circuit is closed and opened. A current constantly flowing through the primary coil would not induce a current in the secondary coil. The induced current is an alternating or to-and-fro current. When the circuit is closed or made, the

induced current in the secondary coil flows in the opposite direction, while the current induced by breaking the circuit flows in the same direction as that of the inducing current. When a bar of soft iron in the centre of the primary coil is suddenly magnetized by closing the circuit and demagnetized by breaking it, currents are induced in the secondary coil. The current induced by magnetization flows in the same direction as that produced by closing the circuit; and that induced by demagnetization as that produced when the circuit was broken. Under such circumstances there would be a double influence acting upon the secondary coil, with consequent increased strength of current. This principle is made use of in the construction of medical batteries for increasing current strength.

As any variation in the strength of the current in a circuit will induce a current in the conductor, a current is induced in the coils of the primary coil. This is termed the extra current. In batteries this is designated as the primary current. It obeys the same laws as to direction that the current induced in the secondary coil does. Accordingly, when the circuit is closed the induced current goes in the opposite direction and they tend to neutralize each other. This current may be consequently disregarded and the extra current considered as flowing in only one direction.

The *strength of the faradic current* increases with the strength of the generating current, the number of windings in the coil, the approximation of the coils, and the suddenness of the change of current strength. When the primary coil is surrounded by the secondary the inducing current is weakened by its induction of a current in the secondary coil. Hence the extra current induced would be weakened, and grows stronger as the secondary coil is removed from the primary. This is utilized in the construction of the *Dubois-Reymond coil*, the extra current being strongest when the secondary coil is slid away from the primary.

High-frequency currents are of special value in the treatment of neuritis, but space does not permit of their discussion here. The sinusoidal current is used principally to cause muscular contractions.

Physiology.—The electrical current **acts upon living tissues** in various ways.

It may cause **electrolysis** or chemical decomposition of the tissues at the electrodes, a property taken advantage of for the destruction of nevi, small tumors, etc.

It possesses the power of **cataphoresis**, *i. e.*, of carrying solutions through the tissues in the direction of the current. By means of this property, cocaine for local anesthesia and other drugs may be introduced into the body at the seat of disease.

It causes a modification of nerve excitability known as **electrotonus.** At and in the vicinity of the *anode* the excitability is lessened—*anelectrotonus;* while at and in the vicinity of the *cathode* the excitability is increased—*catelectrotonus.* Also when anelectrotonus is made to disappear suddenly by breaking the circuit at the anode, the *cathodal increase* occurs and the nerve is thrown into a condition of *catelectrotonus.*

The current also causes **muscular contraction.** These conditions can only be produced by a sudden increase or decrease of electrical excitement in the muscle or nerve supplying it. In other words, by a sudden increase or decrease of current strength. When this is done gradually no contraction results. This being true, the most powerful excitement is caused by the *voltaic alternative.* It is produced by suddenly reversing the current direction, so that a nerve or muscle which had been in a condition of anelectrotonus is thrown into one of catelectrotonus, or from a state of — excitability to one of +, and *vice versa.* By simple closure and opening of the circuit the increase is only from 0 to — excitability and 0 to — excitability, which, of course, is not so great a variation.

The *galvanic current* possesses all of these properties in more marked degree than does the *static* or *faradic.* In these latter the influence of the current is of such brief duration that they are practically only used to cause muscular contractions and to relieve pain, and in the case of the static and high-frequency currents to producee certain metabolic changes.

A *degenerating muscle* loses its power of response to static

electricity first; next, to faradic; then to simple opening and closing of the galvanic circuit, and, finally, to the *voltaic alternative.*

Diagnosis and Prognosis.—Electrical Irritability.—In many diseases of the nervous system the muscles and nerves, when subjected to electrical stimulation, act differently than do normal muscles and nerves. Their *electrical irritability* may differ in two ways—either in *quantity,* meaning a diminution or increase of irritability; or in *quality,* in which, along with the quantitative change, there is one in the *character of the contraction.*

Quantitative Changes.—*Simple increased irritability,* or that in which the muscle or nerve responds to a *weaker* current than normal, is met with most freqeuntly in tetany (p. 489).

Decreased irritability, or that in which a *stronger* current than normal is required to produce contractions, occurs in mild cases of neuritis and sometimes in long-standing central paralyses. These conditions are generally the same for both currents.

EXAMINATION.—To ascertain these facts: if the paralysis exists *on one side only,* the weakest current that will cause contraction of the normal muscle should first be learned; then the difference in the strength of current necessary to produce the same result on the diseased muscles.

If the affection is *bilateral,* the reactions may be similarly compared with the reactions of another normal person; or we judge by experience as to what strength of current is usually normally required.

The **qualitative changes** consist of the *reaction of degeneration* (De R) the *myotonic muscular reaction* and the *myasthenic reaction.* The *former* is found in certain forms of paralysis, and when present always indicates that the causal lesion is situated in the peripheral motor neurone—*i. e.,* either in the motor cells of the medulla or cord, or the axones forming the motor nerves which come from them. The *myotonic* reaction is a symptom of myotonia congenita (p. 491). The myasthenic reaction is a symptom of myasthenia gravis (p. 267) and is characterized by an early exhaustion of the

muscle, especially when stimulated with the faradic current. At first the muscle responds normally, then as the current is continued, in a very short time fails to respond. After a rest the muscle will contract as before.

The **reactions of degeneration** are complete or partial according to the degree of disturbance.

The **complete reaction** consists of (1) a rapid loss of the power of the muscle and its supplying nerve to react to the faradic current; (2) a period of *quantitative* increase followed by a decrease when they are stimulated by the galvanic current; (3) the *modal change, i. e.,* instead of the short, quick, jerky contraction caused by the stimulation of the healthy muscle or nerve by a galvanic current, the contraction is slow, wavy, and sometimes tetanic; and (4) the *serial, change,* when the muscle alone is subjected to galvanic stimulation.

The **serial change** depends upon a difference in the way the muscle reacts to the different poles of the battery. Normally the weakest current that will cause a muscular contraction is when the circuit is *closed* with the cathode *on the muscle* (cathodal closing contraction, CaClC). A little stronger current will cause an anodal closing contraction (AnClC), and so on.

Representing anodal opening contraction by AnOC and cathodal opening contraction by CaOC, the *normal formula* will be CaClC > AnClC > AnOC > CaOC.

The *serial change* in a complete De R consists of an increase of the AnClC and AnOC over the CaClC; so that one formula would read AnClC = AnOC > CaClC > CaOCl. Or this, AnClC > AnOC > CaClC > CaOC.

The **partial** De R is more common than the complete. It may consist of nothing but the loss or diminution of *faradic irritability and the modal change,* the series being normal. Or this may be changed so that AnClC = CaClC. In partial reactions the nerve usually responds normally.

In **examining for the presence** of De R we first use the faradic current as when testing for quantitative changes. Then use the galvanic electrode, placing a large indifferent electrode over the sternum, and the other, a small one, over

the motor point of the muscle (p. 82). We then find the minimum strengths of current necessary to cause CaClC and AnClC. If a meter is at hand, this may be done by noting the number of milliampères required; or if not, by noting that the current which causes CaClC will not cause AnClC, or *vice versa;* or that they are equal.

Another method consists in making the electrode positive and negative, alternately, using the same strength of current and noting the difference, if any, in the intensity of the contractions. In all of these tests there should be a considerable interval (several seconds) between opening and closing the circuit.

The **myotonic reaction** is due to a greatly increased irritability; mild faradic and galvanic currents produce contractions that are tetanic in character, hollows and ridges in the muscle being often produced. In the case of the galvanic current AnClC becomes equal to or greater than CaClC. When the galvanic current is allowed to flow without interruption through a muscle, rhythmical contractions, travelling from the negative to the positive pole occur.

Electricity may sometimes help us in forming a **prognosis.** As the complete De R is only present when extensive damage has been done, the prognosis, as a rule, is more grave than when only the partial De R is present. There are, however, exceptions to this. It is best in those cases where no serial change occurs.

If after ten to fifteen weeks a return of excitability is noticed in any of the places where it had been lost, as a return of faradic contractibility, it is a good prognostic sign. A continuance of increased galvanic irritability, after seven or eight weeks, is a bad prognostic omen.

The fact that stimulation of the auditory nerve, by placing an electrode in front of the ear, will cause nystagmus has been used in diagnosis. This depends on the observation that normally if the anode is the exciting pole the movement will be away from the electrode, and if the cathode is so used it will be toward it. If the auditory nerve is diseased it will be found that less anodal ampères will be required

to cause nystagmus than cathodal. If the nerve reacts normally the trouble is in the labyrinth.[1]

Methods of Application and Therapeutics.—Certain **general rules** should be borne in mind while using electricity. The muscles being treated or tested should be relaxed and in a comfortable position. To cause muscular contraction we should begin with a weak current, gradually increasing its strength until a slight contraction is produced. Violent contractions are harmful. Ten to twelve contractions of each muscle are sufficient. When using strong, galvanic currents always increase and decrease their strength gradually otherwise a painful shock will be caused. To influence a particular spot use a large indifferent electrode, and a small one over the spot. To influence a considerable area two large electrodes may be used. To influence the skin the electrodes should be dry; to reach the tissues under the skin both electrodes should be moist with water or a saline solution. The motor point is a certain spot in each muscle that is more irritable to the action of the current (Figs. 20 to 29). When there is great quantitative decrease contractions too faint to be seen may be often recognized by placing the finger lightly on the muscle.

Treatment.—In the treatment of nervous diseases electricity is used as a *tonic*, either to the general system or to individual muscles and nerves; to increase the sensibility of the skin; as a sedative, for the relief of pain and spasm; and for the introduction of drugs into the diseased parts by utilizing the property of *cataphoresis*. As a tonic we may employ the static, faradic, high-frequency, or galvanic currents.

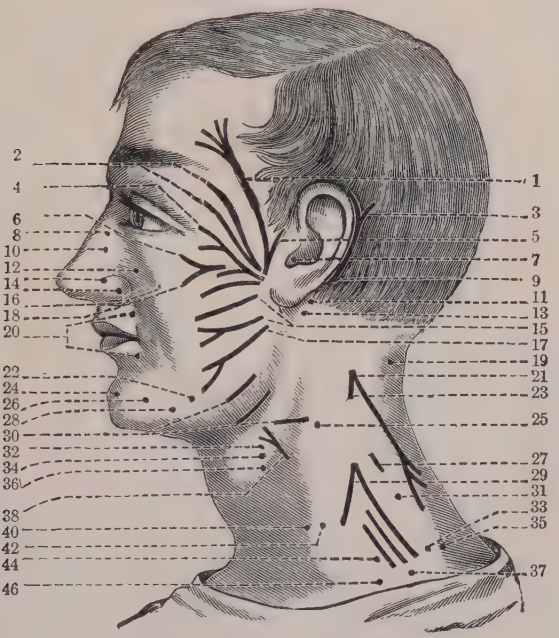
Static electricity is used either as a tonic, sedative, or to produce general metabolic effects. It is also most useful for its psychic influence in the treatment of the hysteria. For the methods of its application the reader is referred to works on electrotherapeutics. The same may be said of high-frequency currents.

The **faradic current**, as a *muscle stimulant*, is only of use in central paralyses, or in peripheral, when the muscle is

[1] G. W. MacKenzie, Archiv. Otologie, 1908.

but slightly degenerated. Either the so-called primary or the secondary current may be used, the latter being somewhat the more powerful.

FIG. 20



A diagram of the motor points of the face, showing the position of the electrodes during electrization of special muscles and nerves. The anode is supposed to be placed in the mastoid fossa, and the cathode in the part indicated upon the diagram. (From von Ziemssen.) 1, occipitofrontalis (ant. belly); 2, corrugator supercilii; 3, occipitofrontalis (post. belly); 4, orbicularis palpebrarum; 5, retrahens et attollens aurum; 6, pyramidalis nasi; 7, facial nerve; 8, lev. lab. sup. et alæ nasi; 9, deep posterior auricular branch of facial nerve; 10, lev. lab. sup. propr.; 11, stylohyoid; 12, dilator naris ant.; 13, digastric; 14, dilator naris post.; 15, buccal branches of facial nerve; 16, zygomat. minor; 17, subcutaneous branch of inferior maxillary nerve; 18, zygomat. major; 19, splenius capitis; 20, orbicularis oris; 21, external branch of spinal accessory nerve; 22, branch of levator menti and dep. ang. oris; 23, sternomastoid; 24, levator menti; 25, sternomastoid; 26, dep. lab. infer.; 27, levator anguli scapulæ; 28, dep. ang. oris; 29, phrenic nerve; 30, subcutaneous nerves of neck; 31, posterior thoracic nerve to rhomboid muscles; 32, sternohyoid; 33, circumflex nerve; 34, omohyoid; 35, posterior thoracic nerve to serratus magnus; 36, sternothyroid; 37, branch of brachial plexus; 38, branch for platysma; 40, sternohyoid; 42, omohyoid; 44, 46, nerves to pectoral muscles.

A convenient method, when using the slowly interrupted current, is to place a good-sized electrode over the nerve supplying the muscle, say, over the brachial plexus if the arm is being treated, while the other electrode, which should be of smaller size, is placed over the motor points (points in the muscles more irritable to the current: Figs. 20 to 29)

Fig. 21

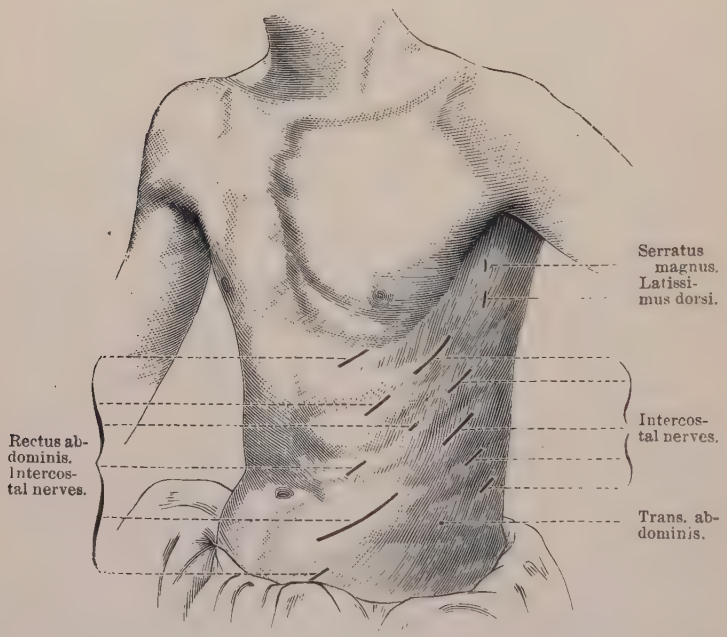


Diagram of the motor points of the trunk. (From von Ziemssen.)

of the muscles being treated. Or, if the rapidly interrupted current only is convenient, the indifferent electrode being in the position above described, we stroke the paralyzed part with the other. This method does not necessitate a knowledge of the position of the motor points. An application every other day is usually often enough.

When used to *stimulate anesthetic skin* we employ the wire

brush and the secondary current rapidly interrupted. The brush is placed over the anesthetic areas, the other being at some indifferent point.

FIG. 22

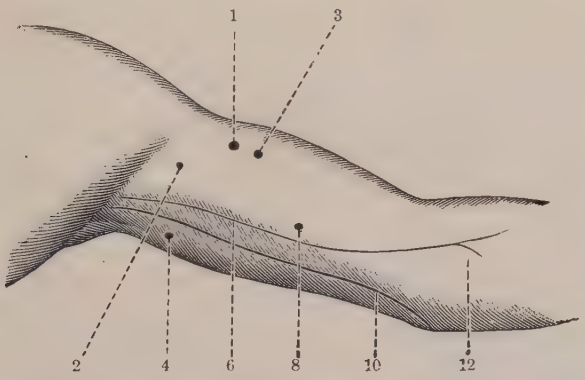
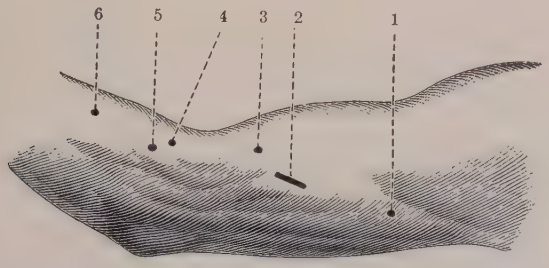


Diagram of the motor points of the arm, under side. (From von Ziemssen.) 1, musculocutaneous nerve; 2, musculocutaneous nerve; 3, biceps; 4, internal head of triceps; 6, median nerve; 8, brachialis anticus; 10, ulnar nerve; 12, branch of median nerve to the pronator trees.

FIG. 23



Motor parts of the arm, outer side. (From von Ziemssen.) 1, external head of triceps; 2, musculospiral nerve; 3, brachialis anticus; 4, supinator longus; 5, extensor carpi radialis longior; 6, extensor carpi radialis brevior.

This should be done daily at least.

For *general tonic purposes* we employ the method known as general faradization. The patient is placed in a recumbent position and all the muscles of the body caused to contract in turn by one of the methods above described. After this

Fig. 24

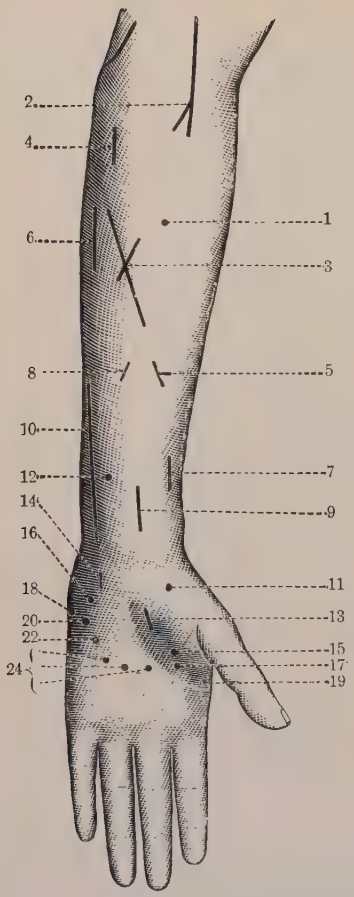


Fig. 25

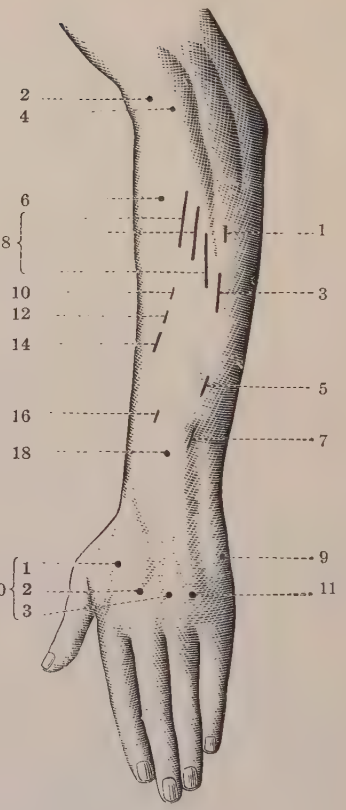


Fig. 24.—Motor points of forearm, inner surface. (From von Ziemssen.) 1, flexor carpi radialis; 2, branch of the median nerve for the pronator teres; 3, flexor profundus digitorum; 4, palmaris longus; 5, flexor sublimis digitorum; 6, flexor carpi ulnaris; 7, flexor longus pollicis; 8, flexor sublimis digitorum (middle and ring fingers); 9, median nerve; 10, ulnar nerve; 11, abductor pollicis; 12, flexor sublimis digitorum (index and little fingers); 13, opponens pollicis; 14, deep branch of ulnar nerve; 15, flexor brevis pollicis; 16, palmaris brevis; 17, adductor pollicis; 18, adductor minimi digiti; 19, lumbricalis (first); 20, flexor brevis minimi digiti; 22, opponens minimi digiti; 24, lumbricales (second, third, and fourth).

Fig. 25.—Motor points of forearm, outer surface. (From von Ziemssen.) 1, extensor carpi ulnaris; 2, supinator longus; 3, extensor minimi digiti; 4, extensor carpi radialis longior; 5, extensor indicis; 6, extensor carpi radialis brevior; 7, extensor secundi internodii pollicis; 8, extensor communis digitorum; 9, abductor minimi digiti; 10, extensor indicis; 11, dorsal interosseus (fourth); 12, extensor indicis and extensor ossis metacarpi pollicis; 14, extensor ossis metacarpi pollicis; 16, extensor primi internodii pollicis; 18, flexor longus pollicis; 20, dorsal interossei.

is done one pole may be placed at the nape of the neck, the other at the feet, and the current rapidly interrupted

Fig. 26

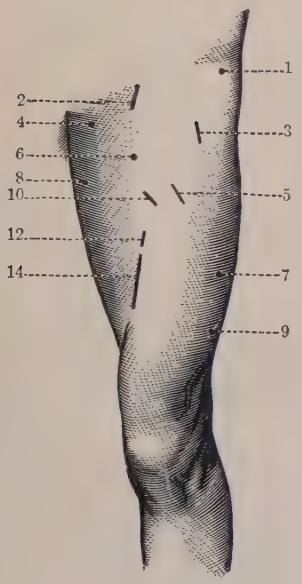
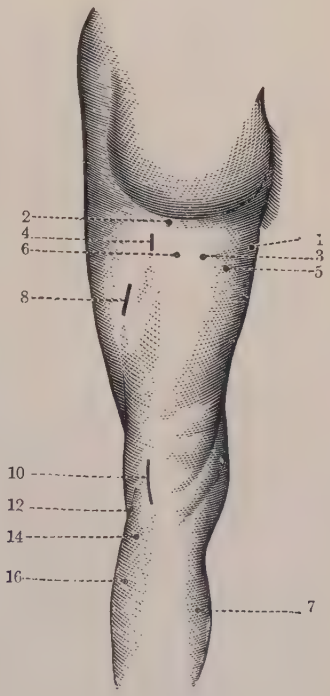


Fig. 27



Motor points of thigh, anterior surface. Posterior surface.

(From von Ziemssen.)

Fig. 26.—1, tensor vaginæ femoris (branch of superior gluteal nerve); 2, anterior crural nerve; 3, tensor vaginæ femoris (branch of crural nerve); 4, obturator nerve; 5, rectus femoris; 6, sartorius; 7, vastus externus; 8, adductor longus; 9, vastus externus; 10, branch of crural nerve to quadriceps extensor cruris; 12, crureus; 14, branch of crural nerve to vastus externus.

Fig. 27.—1, adductor magnus; 2, inferior gluteal nerve for gluteus maximus; 3, semitendinosus; 4, great sciatic nerve; 5, semimembranosus; 6, long head of biceps; 7, gastrocnemius (internal head); 8, short head of biceps; 10, posterior tibial nerve; 12, peroneal nerve; 14, gastrocnemius (external head); 16, soleus.

and made strong enough to produce a general tingling through the body for fifteen or twenty minutes. The treat-

ment should be administered daily, except in women while menstruating.

Fig. 28

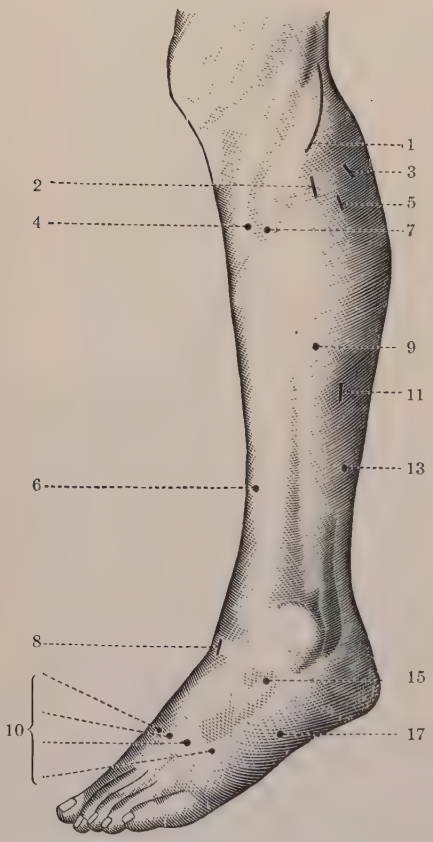
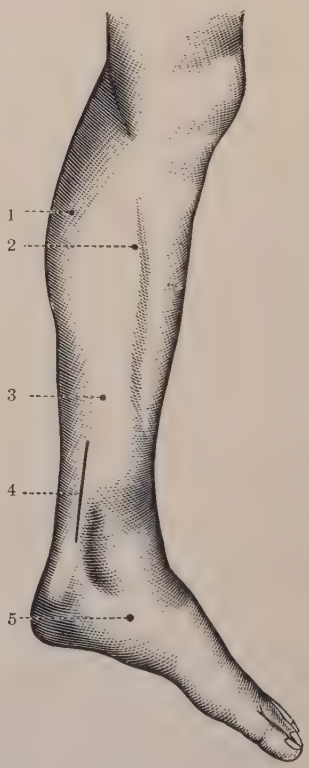


Fig. 29



Motor points of the leg, outer side. Inner side.

(From von Ziemssen.)

Fig. 28.—1, peroneal nerve; 2, peroneus longus; 3, gastrocnemius (external head); 4, tibialis anticus; 5, soleus; 6, extensor longus pollicis; 7, extensor communis digitorum longus; 8, branch of peroneal nerve for extensor brevis digitorum; 9, peroneal brevis; 10, dorsal interossei; 11, soleus; 13, flexor longus pollicis; 15, extensor brevis digitorum; 17, abductor minimi digiti.

Fig. 29.—1, gastrocnemius (internal head); 2, soleus; 3, flexor communis digitorum longus; 4, posterior tibial nerve; 5, abductor pollicis.

The faradic current, especially when rapidly interrupted, exercises considerable psychic influence.

The **galvanic current** when used to influence the *nutrition* of muscles much degenerated, as in severe acute poliomyelitis, is best used by placing the anode at some indifferent point (best over the supplying nerve), while the cathode is rubbed over the muscles without breaking the circuit (labile); or by allowing the cathode to remain stationary at the distal extremity of the limb and the current allowed to flow from ten to fifteen minutes (*stabile*). If we wish to *cause muscular contractions*, the cathode is placed over the motor points and the circuit opened and closed alternately at the cathode. Or the voltaic alternative reversion of the current may be used. Seances when the current is employed labile or stabile may be daily. When contractions are caused, tri-weekly is sufficient.

In some forms of *peripheral paralyses* the galvanic current is the only one of value.

As a *general tonic* it may be used as general galvanization, applied similarly to general faradization.

The **sedative influences** are exercised by each of the forms of current.

For the technique of the application of static and high-frequency currents the reader is referred to works on electrotherapeutics. The *faradic current*, most markedly that from the secondary current, rapidly interrupted, exercises a sedative influence. We employ it by placing an electrode over the painful area. The seance should last five to ten minutes and be repeated frequently. Local spasms may sometimes be relaxed by the same method.

In using the *galvanic current* to relieve pain or spasm the anode is placed over the part to be influenced while the cathode is removed as far away as possible. The current is applied stabile or labile, being gradually increased and very gradually decreased in strength. The current should be as strong as can be borne, and used frequently.

Cataphoresis is used in neurology principally in the treatment of *neuralgia*. If a solution of cocaine, 10 per cent., is used to moisten the anode which is placed over the painful

nerve, some of the cocaine will penetrate the skin and directly affect the painful part. Special electrodes, the best of which is Peterson's, have been devised for the employment of cataphoresis.

MASSAGE

Massage is the name given to a number of manipulations of the body, which possess marked value in the treatment of nervous diseases as well as of others.

Effects.—Massage properly applied stimulates the flow of the lymph and venous currents; increases temporarily the number of red blood corpuscles; accelerates the heart's action; stimulates the motor nerve endings and muscular contractility; and, according to the method used, exercises either a stimulant or a sedative action upon the sensory nerves.

Indications.—It is therefore of value in various forms of neurasthenia, hysteria, motor paralysis, anesthesia, headache, neuralgia, function spasms, insomnia, and constipation. It should not be employed where there is a diseased heart or bloodvessels.

To *administer massage* properly requires special training. The **movements** consist of *effleurage* or gentle stroking; *massage á friction*, consisting of vigorous strokes with one hand and strong circular and to-and-fro friction with the other; *pétrissage* or kneading; and *tapotement* or striking with the hands or ends of the fingers. Passive movements are frequently combined with these.

Effleurage and tapotement are most useful in treating painful conditions.

Muscular Movements.—Regular muscular movements, according to a fixed schedule, as first used by S. Weir Mitchell, but since elaborated by Frenkel, are of value in the treatment of muscular incoördination. The exercises should be performed with care and precision twice daily.

The following schedule, based upon that of Frenkel[1] and Hirschberg, is recommended by Dana:

[1] See Tabetic Ataxia, by H. S. Frenkel.

Exercises for the Hands and Arms.—1. Sit in front of a table, place the hand upon it, then elevate each finger as far as possible. Then raising the hand slightly, extend and then flex each finger and thumb as far as possible. Do this first with the right, then with the left. Repeat once.

2. With the hand extended on the table, abduct the thumb and then each finger separately as far as possible. Repeat three times.

3. Touch with the end of the thumb each finger tip separately and exactly. Then touch the middle of each phalanx of each of the four fingers with the tip of the thumb. Repeat three times.

4. Place the hand in the position of piano playing and elevate the thumb and fingers in succession, bringing them down again, as in striking the keys of the piano. Do this twenty times with the right hand and the same with the left.

5. Sit at a table with a large sheet of paper and pencil. Make four dots in the four corners of the paper and one in the centre. Draw lines from corner dots to centre dot with right hand; same with the left.

6. Draw another set of lines parallel to the first with the right hand; same with the left.

7. Throw ten pennies upon the paper. Pick them up and place them in a single pile with the right hand; then with the left. Repeat twice.

8. Spread the pennies about on the table. Touch each one slowly and exactly with the forefinger of the right hand; then with the forefinger of the left.

9. Place an ordinary solitaire board on the table, with the marbles in the groove around the holes. Put the marbles in their places with the right hand; same with the left hand. Patient may, with advantage, practise the game for the purpose of steadying his hands.

10. Take an ordinary fox-and-geese board with holes and pegs, and, beginning at one corner, place the pegs in the holes, one after the other, using first the right hand and then the left.

Exercises for the Body and Lower Limbs.—1. Sit in a chair. Rise slowly to the erect position without help from cane or arms of chair. Sit down slowly in the same way. Repeat once.

2. Stand with cane, feet together, advance the left foot and return it; same with the right. Repeat three times.

3. Walk ten steps with cane, slowly. Walk backward five steps with cane, slowly.

4. Stand without cane, feet a little spread out, hands on hips. In this position flex the knees, and stoop slowly down as far as possible; rise slowly. Repeat twice.

5. Stand erect, carry left foot behind and bring it back to its place; the same with the right. Repeat three times.

6. Walk twenty steps, as in exercise No. 3; then walk backward five steps.

7. Repeat No. 2 without cane.

8. Stand without cane, heels together, hands on hips. Stand in this way until you can count twenty. Increase the duration each day by five, until you can stand in this way while one hundred is being counted.

9. Stand without cane, feet spread apart; raise the arms up from the sides until they meet above the head. Repeat this three times. With the arms raised above the head, carry them forward and downward, bending with the body until the tips of the fingers come as near the floor as they can be safely carried.

10. Stand without cane, feet spread apart, hands on hips; flex to trunk forward, then to the left, then backward, then to the right, making a circle with the head. Repeat three times.

11. Do No. 9 with heels together.

12. Do No. 10 with heels together.

13. Walk along a fixed line, such as a seam on the carpet, with cane, placing the feet carefully on the line each time. Walk a distance of at least fifteen feet. Repeat twice.

14. Do the same without cane.

15. Stand erect with cane; describe a circle on the floor with the toe of the right foot. Same with the left. Repeat twice. Between the fifth and sixth exercise the patient should rest for a few moments.

HYDROTHERAPEUTICS

Water may be used either as a *tonic* or as a *sedative.*

As a **tonic** we employ cold plunges; shower baths; various forms of douches, as Charcot's and the Scottish; cold sitz bath, salt baths, either sea or artificial, and short cold packs. These all have a stimulating and tonic effect. In giving them, especially to weak people, it is best to begin with warm water and gradually lower the temperature. Showers and douches are the most stimulating. A reaction should always be obtained by vigorous rubbing afterward.

For the *cold plunge* the patient jumps into a tub of water at a temperature of 60° to 70°, and at once jumps out. He should then be rubbed vigorously.

The *douche* is probably the best means of obtaining tonic effects. It is usually given with a pressure of 15 to 20 pounds to the square inch, but at first a less pressure is often preferable. The temperature of the water should vary from 80° to 45°. The higher temperature should be used for the first treatments, when, if these are borne well, it is gradually reduced, 55° to 65° being the average temperature used. A good reaction should follow the treatment, which is only continued for a few minutes. Before giving the douche, wrapping the patient in a hot blanket or placing him for a few minutes in a hot-air box adds to the efficacy of the treatment.

If a douche cannot be obtained a cold ablution may be substituted. This consists in standing the patient in warm water, and by the hand or a large sponge, splashing water at a temperature of 50° to 60° over him for a few moments, this to be followed by vigorous rubbing with a warm towel. The patient, after these procedures, should experience a feeling of warmth and well-being, otherwise he is not reacting properly.

The *shower* or *rain bath* consists in allowing water to fall on the body from a height, for one or two minutes, while the feet are in warm water. Frictions of the body should be kept up during this process.

A Charcot douche is given by directing a solid stream of water with force upon the back of the patient.

By the *Scottish douche* we mean alternating a cold douche with a warm or a hot one.

Cold packs are given by wringing a sheet out in cold water (50° to 80°), wrapping it about the patient for a few moments, when it is removed and the patient put to bed and rubbed.

An *artificial salt bath* may be made by putting twenty-five pounds of salt in thirty gallons of water (2 per cent.). It may be warm or cold. Arteriosclerosis or great weakness of the circulation contraindicates cold applications.

To obtain **sedative effects** we may use the lukewarm bath, wet pack, hot sitz baths, hot compresses, and drip sheet.

The *lukewarm bath* is given at a temperature of 95° to 98° for ten to twenty minutes.

The *continuous bath* is sometimes employed in delirious and maniacal states. A special arrangement must be used to keep the water at an even temperature (100° F.) and for the head of the patient to rest upon. The bath may be continued for hours, the patient eating and sleeping there. An attendant must be constantly present.

To give a *wet pack* we spread a large thick blanket upon the bed; upon this is laid a sheet wrung out in water at a temperature of 60° to 70°. The nude patient lies upon this and the sheet is wrapped smoothly about him, not including the head and feet. The sheet must be carried between the legs and brought evenly in contact with the body. The blanket is then folded over him, with others added if desired. Hot-water bottles may be placed at the feet, and cold compresses to the head. The patient lies in this for one-half hour and is then rubbed off.

The *drip sheet.* Have a basin of water at 65°. Put in the basin a sheet. The patient stands in comfortably hot water. Have ready a large soft towel and iced water. Wring out the towel in this and wrap it around the head and back of the neck. Standing in front of the patient, the sheet is seized by the two corners and thrown about the patient, who holds it at the neck. It is then smoothed out over the body. It is then dropped and the patient made to lie

down on a blanket, which is wrapped about him. Dry thoroughly with coarse towels, wrap in a dry blanket for a time, then put to bed. The water should be gradually cooled day by day to 55°.

The bath, pack, and drip sheet are valuable remedies for insomnia.

A *hot sitz bath* consists of the patient's sitting in water at a temperature of 100° to 125° from twenty to thirty minutes.

Hot compresses are often used for the relief of local pains and congestions. Many other methods of using water, therapeutically may be found in works on hydrotherapeutics.

THE REST TREATMENT

This method of treatment, devised by S. Weir Mitchell, is of special value in neurasthenia and hysteria.

Its **essential features** are isolation, absolute rest, diet, massage, electricity, and the personal influence of a good nurse.

Isolation from the patient's family and former surroundings is essential. The *diet*, if the digestion is bad, should be entirely of milk. Otherwise easily digested, solid food may alternate with it.

Rest must be absolute in severe cases; even sitting up in bed should not be allowed. The duration of the treatment should be from four to eight weeks.

The **following schedule**, as given by Dr. J. K. Mitchell, will illustrate the method:

7.00 A.M.: Cocoa; cool sponge bath, with rough rub and toilet for the day.
8.00 A.M.: Milk. Breakfast. Rest for one hour.
10.00 A.M.: 8 oz. peptonized milk.
11.00 A.M.: Massage.
12.00 M.: Milk or soup; reading aloud by the nurse.
1.30 P.M.: Dinner; rest one hour.
3.30 P.M.: 8 oz. peptonized milk.
4.00 P.M.: Electricity (general faradization).

6.00 P.M.: Supper, with milk.
8.00 P.M.: Reading aloud by nurse for one-half hour.
9.00 P.M.: Light rubbing by nurse with drip sheet.

Tonic after meals. 8 oz. peptonized milk with biscuit at bedtime, and a glass of milk during the night if desired.

This may be modified in various ways according to the symptoms. If a mild case, the patient may be allowed to sit up for a few hours or even go out for a drive.

LUMBAR PUNCTURE

Valuable diagnostic aid, and at times therapeutic results, may be obtained in certain diseases of the nervous system by withdrawal and examination of the cerebrospinal fluid. The technique of the operation is as follows: The puncture

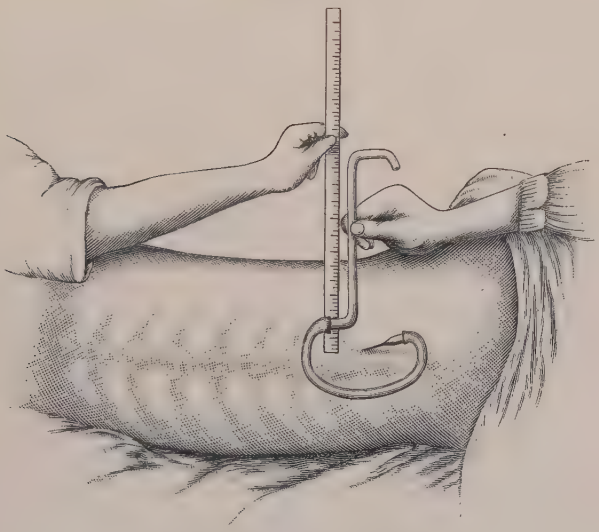
FIG. 30

Lumbar puncture; introducing the trocar. (Musser.)

should be made between the third and fourth lumbar vertebræ in the median line in children, but about one-half inch to one side in an adult. This point may be determined by drawing a line through the highest point of the crests of the ilia which will cross the fourth lumbar interspace. A further aid is the fact that the spinous process of the fifth lumbar vertebræ is more prominent than the spinous

processes of the sacrum. The patient should preferably be placed on the left side with the thighs flexed upon the abdomen and the head bent forward as far as possible. The point to be punctured should then be marked and the back sterilized in the usual way (this of course also applies to the instruments and hands of the operator). The needle, which may be an antitoxin needle, but preferably one made for the purpose, is grasped in the right hand, the point placed over the mark on the skin and held at an angle of

FIG. 31



Measuring the pressure. (Quincke.)

45 degrees with the surface of the back (Fig. 30) inclined slightly toward the median line. It should then be thrust forward slowly and steadily. When it has been introduced from two and one-half to three and one-half inches it may be assumed that it has entered the canal and the mandril may be withdrawn. As soon as the fluid begins to flow the manometer may be attached (Fig. 31) and the pressure determined. This, however, can be very well determined

by sight. High pressure being indicated by a spurt and steady stream, moderate by a slow steady stream, and low if the fluid comes drop by drop. Normal pressure is indicated by a flow of 60 drops per minute (Mott). This varies somewhat if the observation is made with the patient sitting or recumbent, being stronger in the former. If measured with a manometer, above 40 mm. of mercury in a recumbent person is abnormal. After this the fluid is allowed to flow into a receptacle until it begins to drop slowly, when the needle is withdrawn and the puncture sealed with cotton and collodion.

Fluid may fail to flow owing to the needle striking the arch of the vertebræ, when it should be partly withdrawn and reintroduced in a different direction. It may become clogged with gelatinous or purulent exudate. The membranes may be pushed in front of the end of the needle or it may be in contact with a nerve root, which can usually be dislodged by reinserting the mandril. The fluid may be bloody, owing to the wounding of a vein, which may lead to faulty conclusions. The patient may become dizzy and nauseated, and complain of headache; if so, the operation should at once cease.

The points to be determined in studying the fluid are:

1. The pressure under which it is obtained.
2. Its color and appearance after standing.
3. The bacteriological findings.
4. Cytodiagnosis.
5. Chemical examination.
6. Cryoscopy of the fluid.

The first five are the most important.

1. High pressure indicates irritation due to the presence of some toxic substance in the blood, as uremia, or some inflammatory process, as meningitis. Heightened pressure may also occur in acute hydrocephalus, brain tumor, and brain abscess. It is apt to be specially high in tubercular and epidemic cerebrospinal meningitis. There may be a moderate increase in the functional neuroses.

2. The admixture of blood is the principal fact determined from the color. In chronic meningitis it may be of a greenish-

yellow color, and in acute meningitis it may be turbid or purulent. Normally it is clear like water, the sp. gr. being 1004 to 1007, and boiling with Fehling's solution shows the sugar reaction. It is usually clear in tubercular meningitis. Coagula form in certain abnormal conditions if the fluid is allowed to stand in a tube. This is specially seen in inflammatory conditions and if hemorrhage has occurred into the subarachnoid space. In tubercular meningitis the fluid is usually quite clear, but a delicate web-like coagulum will form, if it stands for six hours or more.

3. The bacterial findings are of importance. The various germs causing meningitis excepting tubercular being found according to the cause of the inflammation.

4. Normally the fluid contains few if any leukocytes; if these are plentiful (more than 7 per c.mm., known as pleocytosis) it is evidence of meningeal irritation. Methods have been devised for staining the cells and a special slide devised for counting them (Fuchs and Rosenthal[1]). Differential counts are important, as in purulent and epidemic cerebrospinal meningitis polynuclear cells predominate, while in tubercular meningitis lymphocytes are more numerous, except in the very early stage when more polynuclear cells may be seen. Lymphocytes are the predominant form of cell in tabes, paresis, and cerebrospinal syphilis. Plasma cells are found principally in paresis. In cases of suspected syphilis the Wassermann test should be performed with the cerebrospinal fluid as well as the blood[2] (p. 412).

5. A slight amount of proteid in the form of globulin is normally present (not more than 0.05 per cent.). In inflammatory affections of the meninges, and in affections of the cerebrospinal system due to syphilis, it is increased. Various tests have been devised to show its presence, as the Nonne-Apelt, Ross-Jones, and Noguchi butyric acid.

The Ross-Jones test is performed as Heller's nitric acid

[1] Henderson, Rev. Neurol. and Psychiat., March, 1912, p. 109.

[2] It is important to remember that the Wassermann reaction alone does not necessarily indicate syphilis of the nervous system, as it may be due to visceral syphilis. All of the four methods of examination above mentioned should be employed. Antisyphilitic medication within six months may cause a negative reaction.

test for albuminuria is done. 2 c.c. of a saturated solution of ammonium sulphate are placed in a test-tube and 1 c.c. of cerebrospinal fluid is allowed to gently flow down the tube. The formation of a ring at the junction of the two fluids constitutes a positive reaction (Noune's Phase I).

A small amount of sugar is also present normally which disappears in meningitis except tubercular. Its presence may differentiate cerebrospinal rhinorrhea from rhinitis.

While lumbar puncture has been used as a therapeutic measure in most all diseases of the brain and cord, its most valuable results are obtained in relieving pressure when there is an excess of cerebrospinal fluid and in removing with the fluid the toxic agents which produce the disease. It is therefore of most value in meningitis, cerebral or spinal, and has also been of service in uremia.

Lumbar puncture is not a procedure to be used indiscriminately and for the sake of doing it. Death has resulted from it. It is specially contraindicated when arteriosclerosis exists, and in all diseases of the nervous system without pressure symptoms arising from the cerebrospinal fluid. As a diagnostic measure it should only be used when the diagnosis is in doubt. In brain tumor it has caused serious symptoms. Suction to withdraw the fluid should never be employed.[1]

[1] For a full discussion of the procedure see the article by S. J. Kopetzky in the Amer. Jour. Med. Sci., April, 1906, p. 648, and Ebright, Jour. Amer. Med. Assoc., November 7, 1908, p. 1566. For the characteristics and methods of examination of the fluid and their diagnostic significance see Kaplan and Casamajor, Archives of Internal Medicine, February 15, 1912, p. 262.

CHAPTER V

SYMPTOMATIC DISORDERS

THE disorders about to be described always occur as symptoms; but as symptoms they stand out so prominently as to be often spoken of as diseases *per se*.

HEADACHE (CEPHALALGIA)

Definition.—A diffuse pain, which may be paroxysmal or continuous, affecting different parts of the head, and not confined to the seat of any particular nerve.

Etiology.—The *causes* of headache are many. They are conveniently expressed in the *following summary*, modified from that of J. C. Wilson:

1. **Reflex Irritation**—viz., ocular (eye-strain or inflammatory disturbances); nasal; pharyngeal; disease of accessory sinuses; auditory; decayed teeth; reproductive organs (especially female); thoracic and abdominal viscera.

2. **Toxemic.**

A. Infections, as acute infectious diseases; malaria; syphilis before secondary symptoms appear.

B. Incomplete or perverted *physiological chemical processes,* or the defective elimination of waste: as uremia; diabetes; gout; lithemia; rheumatism; gastrohepatic derangements; constipation; exophthalmic goitre.

C. Action of drugs and poisons:

a. Acute: nitrites; quinine; opium; alcohol; carbon dioxide, etc.

b. Chronic: lead; tobacco; alcohol; opium; tea; coffee.

3. **Circulatory Disturbances:**

A. Passive congestion, as by posture; tight clothing about the neck; pressure on veins by tumors; disease of right side

of the heart; emphysema and other conditions of the lungs which prevent the free circulation of the blood.

B. Active hyperemia from excessive physical or mental strain; early stage of acute meningitis.

C. Anemia following loss of blood or the idiopathic anemias, especially chlorosis; diseases of the heart which prevent the blood from reaching the brain, as aortic stenosis, fatty degeneration.

D. Changes in the arteries from endarteritis; arteriosclerosis.

4. **Neuroses,** as epilepsy; hysteria; neurasthenia.

5. **Organic disease of the brain** or its membranes, viz., meningitis, syphilitic, traumatic, or otherwise; encephalitis; abscess; tumor; aneurysm.

6. **Caries** of the cranial bones.

7. **Indurative or muscular,** due according to Edinger to the presence of rheumatic nodules in the muscles of the scalp and neck.[1]

8. **Migraine.** (See p. 105.)

The *most common* of these causes are eye-strain, gastrohepatic derangements, constipation, alcohol, uterine disorders, anemia, neurasthenia, indurative and febrile affections. Several of these may coexist.

Pathology.—Of the exact mechanism of the production of a headache we know nothing. The brain substance itself is apparently insensitive to direct irritation, but processes which irritate sensory nerves in any part of their course may be referred to the cortical centres of these nerves and appreciated as *pain.* The dura is sensitive to direct irritation. Most headaches of organic origin are due to irritation of that membrane. While the pain in the majority of headaches appears deep seated, in some cases it is referred to the scalp, and these are usually associated with tenderness; such are usually due to either rheumatism (indurative) or syphilis.

[1] Müller (Deutsch. Zeits. f. Nervenheilk, Band xl, Heft 3 and 4) believes that they are due to an increased tension and shortening of the muscles and calls them "muscular headaches."

The origin of some headaches is possibly explained by the arrangement of the nerve supply of the dura and scalp. The supply of the falx, tentorium, and anterior three-fourths of the dura is derived from the fifth nerve; the posterior fourth from the sensory fibers of the vagus. The scalp as far back as the vertex is also supplied by the fifth, the remainder deriving its supply from the posterior branches of the upper four cervical nerves.

The sensory (descending) root of the fifth nerve is in close relation with the origin of the cervical branches in the cord and of the cranial nerves in the medulla; the regions of the dura supplied, respectively, by the fifth and vagus overlap; the vagus furnishes part of the nerve supply of the viscera. In addition the pia (according to Gray) is supplied with branches from the third, fifth, sixth, seventh, ninth, tenth, and eleventh cranial nerves, the posterior branch of the first cervical and the sympathetic. These relations readily account for the fact that headache is so commonly caused by irritative conditions in other organs.

Headache is also produced by anything which causes increased *blood pressure;* all headaches, excepting those due to impoverishment of the blood supply, are made worse by sudden lowering of the head.

Symptoms.—The pain in headache often differs in character and location, according to the cause. These facts, while not absolute, may be of service in diagnosis. It may be pulsating or throbbing; dull and heavy; constricting or pressing; hot, burning or sore; boring or sharp.

In *location* it may be frontal, occipital, parietal, and temporal, vertical, diffused, or combinations of these.

A pulsating or throbbing pain, situated either in the vertex or diffused, is characteristic of that due to circulatory disturbances. The dull, heavy pain, often frontal, is frequently found in toxic headaches. The constricting or sense-of-pressure type is common in neurasthenia. Patients often describe this as a tight band being drawn about the head. Headaches of a neurasthenic type may be present in children owing to overwork at school. The hot, burning pain, usually vertical, is common in headaches due to

anemia. The sharp boring variety is found in the hysterical and neurotic. It is often described as if a nail were being driven into the head, and is then called the clavus hystericus.

Headache, caused by hypermetropia, astigmatism, or lack of muscle balance, is aggravated by using the eyes, and is associated with other symptoms of asthenopia; is better in the morning, and situated either just over the orbits or in the occiput, or both. Headache is also caused by deep inflammatory conditions, as iritis, ulcer, etc., and by glaucoma. The pain in the latter is usually temporal.

Disease of the throat, nose, or accessory sinuses are common causes, and these parts should always be investigated in all chronic headaches. The pain in the latter two conditions is frontal, often extending to the occiput, and comes on an hour or so after rising and disappears toward evening. It is aggravated by lowering the head or jarring it in any way.

The syphilitic and organic headache is apt to be markedly aggravated at night, being so severe as to prevent sleep; functional headaches rarely do this.

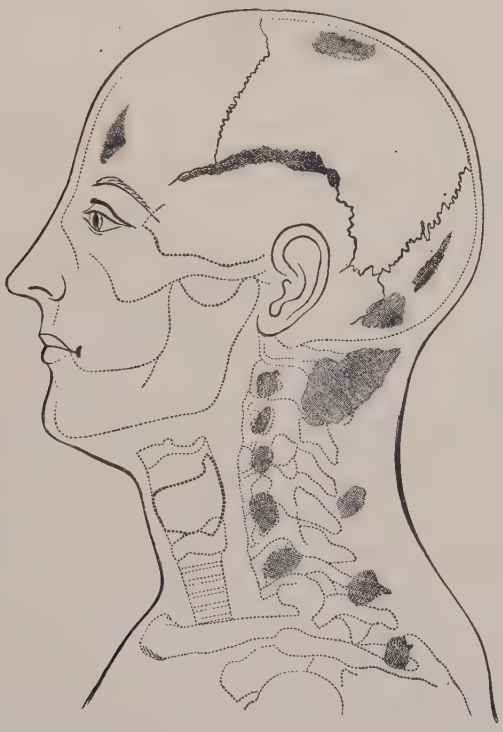
Indurative headaches[1] (p. 100) are exceedingly common. Edinger believes they are more so than those due to any other cause. They are usually due to either exposure to cold winds, washing the hair, or the rheumatic or gouty diathesis. The character of the pain closely resembles that of migraine (p. 105). Examination shows areas of tenderness with swelling and induration where the muscles of the neck are inserted into the skull. Similar areas may also be found lower down in the neck (Fig. 32). This tenderness can also be found in the intervals between attacks of pain. Edinger states that "*the examination of the muscles should never be neglected in any case of headache.*"

Symptoms which may *accompany headache* are vertigo, nausea, and somnolence. The usual symptoms which accompany the condition causing the headache would also be present, and their presence would, of course, be of assistance in ascertaining the cause of the pain. In examining a

[1] Jour. Amer. Med. Assoc., April 24, 1909, p. 1316.

case of chronic headache all of the causes detailed above must be borne in mind, and careful inquiry made as to the condition of the eyes; nose; throat; teeth; pelvic organs, if a woman; heart; bloodvessels; blood; digestive organs; habits as regards alcohol, tobacco, etc., and the possible existence of neurasthenia, hysteria, and organic disease of the brain or membranes.

Fig. 32



The points upon which "indurations" are most frequently formed. (Edinger.[1])

Diagnosis.—Headache must be distinguished from migraine, of which headache is a symptom, and neuralgia. The pain of headache is usually bilateral, and is more or less

[1] Diseases of the Nervous System, edited by Archibald Church.

persistent; that of migraine is paroxysmal and unilateral, and is associated with other sensory disturbances; it passes off, leaving the patient feeling better than before, to return again at more or less regular intervals (p. 105). The pain of neuralgia is shooting and limited to the course of a nerve, along which there are often tender points (points of Valleix).

Having determined that headache is the condition present, we then ascertain to which of the causes mentioned above it is due.

Treatment.—The treatment of headache is essentially that of its cause. Various measures, however, are often required for the immediate relief of the pain. Antipyrin, gr. v–x, either alone or combined with the bromides and aromatic spirit of ammonia, given every hour until two or three doses are taken; phenacetin, gr. v–x, repeated in an hour if necessary; combinations of acetanilide, gr. ij–iij, and caffeine, gr. ss–j, are all valuable. Caffeine and its salts are especially beneficial in the neurasthenic headache. Combinations of caffeine and ammonium salicylate may prove useful. Fluidextract of ergot in doses of a teaspoonful, repeated hourly until three doses have been given, is recommended in cases in which there is vascular engorgement. In rheumatic headaches, in addition to the use of salicylates, massage of the scalp is of service.

In cases due to constipation and gastrohepatic disturbance small doses of calomel and bicarbonate of sodium should be given, to be followed by general treatment for those disorders to prevent return.

Local measures are sometimes of service; mustard plasters to the nape of the neck, or a hot mustard foot bath with cold to the head is useful in congestive headaches; stimulating liniments; menthol, either the pencil or 20 per cent. solution in alcohol; a tight bandage about the head may all prove useful.

The headache of arteriosclerosis is often benefited by the use of nitroglycerin. Cases are reported of headache, the cause of which could not be determined, and in which, after all other measures had failed, the removal of a button of bone by the trephine has afforded relief. These measures

should all be associated with rest, quiet, and light diet. Other measures for the relief of this symptom will be found under treatment of the various conditions which cause it.

The remedy for indurative headache is massage of the neck muscles and indurated areas, beginning with gentle stroking toward the body and gradually becoming more vigorous. Salicylates and at times iodides internally should be combined with this. The constant galvanic current, the anode over the painful areas may also be employed, and vibration can be used instead of massage, unless the patient has marked arteriosclerosis.

MIGRAINE

Synonyms.—Hemicrania; sick headache; megrim.

Definition.—Migraine is more than headache, but may be conveniently considered here. It may be defined as a constitutional neurosis, associated with violent paroxysms of pain, usually unilateral and confined to the course of the fifth nerve, and frequently accompanied by other sensory disorders and with nausea or vomiting, vasomotor disturbances and mental depression.

Etiology.—Migraine is a disease of the first half of life, very few cases beginning after thirty. Heredity, either direct or indirect, is the most important predisposing factor. When *direct*, members of the family for several generations back have been afflicted. When *indirect*, they have been sufferers from other neuroses, especially neuralgia or epilepsy. Frequently other members of the same generation are sufferers either from migraine or one of the neuroses above mentioned. The gouty diathesis appears to have some influence; a father may be gouty and the son have migraine.

In some cases, when the disease appears late, influences that depress or weaken the nervous system, as excessive brain work, anemia, and reflex irritations, seem to act as exciting causes. In those who are already sufferers, an attack may be precipitated by fatigue, excitement, digestive disorders, and eye-strain either by overuse or watching moving objects.

Pathology.—Migraine possesses no morbid anatomy. It is thought by some to be due to periodical "discharges" from the sensory centres of the cortex, similar to the discharges which occur from the motor centres in epilepsy. A largely accepted view is that a vasomotor spasm occurs followed by dilatation, causing successively anemia and hyperemia, which, by acting on the nervous tissues, cause the headache and various temporary diminution of functions (paralysis, hemianopsia, etc.) which occur.

Symptoms.—In a typical attack the patient may or may not have, for a day or so, *prodromes* as a feeling of heaviness in the head and somnolence. Usually the onset is sudden. A bright spot suddenly appears on one side of the visual field, which enlarges, becoming darker in the centre, and changing from round to angular and zig-zag shapes (fortification spectra). In ten to thirty minutes these disappear, pallor of the face appears, and the pain begins usually in one temple, spreading until one side of the head is involved. The pain is increased by noise, light, or stooping, but feels better when a recumbent posture is maintained. In a few hours nausea is felt, which in a greater or less length of time culminates in the vomiting of first the stomach contents, then mucus and bile, the bile being due to retrostaltic action of the stomach. This is succeeded by sleep and relief from pain. The attack usually lasts about twenty-four hours, but in severe cases it may be longer.

The pain, while usually unilateral, may be bilateral. Instead of the form of *sensory* disturbance above described, figures of men and animals and other forms of visual hallucination may be present. In other cases there is first noticed dimness of sight on one side, which increases until half of each field is blind (homonymous hemianopsia). Rarely, instead of visual, disturbances of other special senses occur, as one-sided deafness, tinnitus, loss of taste or subjective sensations of taste. Tingling of the arm followed by anesthesia, or *vice versa*, precedes the pain in rare instances. Another rare symptom, occurring when the pain is left-sided, is a transient aphasia. This may be either sensory, motor, or paraphasia. This symptom may be associated

with transient right-sided tingling, numbness, or motor weakness of the arm; more rarely of the leg. Paralysis of the muscles which move the eyeball may precede the pain or come on during the attack (ophthalmoplegic migraine, p. 145). There may also be mental depression, restlessness, and either temporary loss or increase of memory.

The vasomotor symptoms are usually pallor of the face and coldness of the extremities (angiospastic type); more rarely there is flushing with sweating, sometimes unilateral (angioparalytic type), or these conditions may alternate. In children fever is often present.

The attack does not always follow the type above described. Very rarely it may consist of some of the various sensory disturbances described, with very little or no pain; or of that which is the most common of all forms, *i. e.*, headache associated with vasomotor disturbance, nausea, and vomiting.

These attacks occur from two weeks to a month apart. Patients with migraine sometimes suffer from vertigo in the intervals.

Diagnosis.—The distinctions between migraine, headache, and neuralgia have already been mentioned (p. 103). Those cases in which there are disturbances of special senses without headache closely resemble minor epilepsy with a visual or other special sense aura. The epileptic attack is of very brief duration—a few seconds—while the disturbance in migraine lasts twenty to thirty minutes. Epilepsy and migraine may coexist.

Prognosis.—As regards life the prognosis is good. As regards ultimate recovery it is not so good; but there is a tendency for the attacks to diminish in the second half of life (the menopause in women). The prognosis is better when a history of heredity in which this or other neurosis has been present is not obtained. It is also more favorable when the disease is of recent development. In the majority of cases much can be done by appropriate measures to ameliorate the symptoms.

Treatment.—*Prophylaxis* is of importance. The children of families in which this or other neurosis exists should be

carefully watched. All possible sources of reflex irritation (eye, nose, digestive organs) should be avoided or removed. Excessive mental and physical strain must be avoided, and an outdoor and regular life encouraged.

Treatment may be divided into that of the attack and that of the disease.

For the *attack*, rest in bed in a darkened room as soon as the premonitory symptoms appear must be enjoined. If possible the stomach should be washed out with hot water (105° or more). If this cannot be done, large quantities of hot water should be drunk. After this a saline cathartic, as a teaspoonful or two of Carlsbad salts, should be administered, the action of which may be hastened by a hot soap-and-water enema. Combined with these measures antipyrin, phenacetin, acetanilide, caffeine, the salicylates, and ergot, and the various local measures may all be tried, as recommended for headache. In addition, mild galvanic currents applied to the head are sometimes of benefit. The angiospastic type may often be relieved by nitroglycerin in full doses.

In treating the *disease* all possible sources of reflex irritation must be removed; the gastro-intestinal tract put in good condition; and the diet must be simple, fermentative articles being avoided, and a very scant allowance of all flesh foods permitted. Of *drugs*, long-continued courses of cannabis indica to point of toleration are most successful. This may be combined with arsenic; or, if there is a gouty diathesis, with ammonium salicylate or some of the salts of lithia. Either phosphate of sodium or Rochelle salt in water before breakfast is also of service. Combined with the use of these drugs should be the measures detailed under *prophylaxis*.

NEURALGIA

Definition.—Neuralgia is a functional disease of the sensory fibers of the nerve trunks, and is characterized by pain (Sinkler).

Forms.—Neuralgias may be *idiopathic—i. e.*, developed "spontaneously;" or *symptomatic—i. e.*, due to some known

cause. According to their location they are divided into trigeminal or neuralgia of the fifth nerve; sciatic; intercostal; cervicoöccipital; brachial; lumbo-abdominal; crural; and visceral. Trigeminal, sciatic, and intercostal are by far the most frequent.

Etiology.—Neuralgia is a disease of adults, being rare in childhood and old age. Those possessing a neurotic constitution or a gouty or rheumatic diathesis are especially prone to it. It is more common in cold and damp climates, and in winter than in summer. The exciting causes of symptomatic neuralgia are exposure to cold; toxemias, as la grippe; uremia; gout; rheumatism; malaria; diabetes; lead and other metallic poisons; anemia; arteriosclerosis; debility from any cause; reflex irritations, as eye-strain, carious teeth, gastro-intestinal derangements; neurasthenia and hysteria.

Pathology.—In many cases of so-called neuralgia there is really a low-grade neuritis. This is especially true in the sciatic form. In those cases due to toxemias the pain is probably due to the irritant action of the poison. That due to anemia is often spoken of as "the cry of the nerve for more blood."

Cases[1] occurring late in life have been found to be associated with arteriosclerosis. In a number of Gasserian ganglia and the nerve divisions which were removed by Keen for tic douloureux, Spiller found there were in most instances marked evidences of degeneration of the nerve fibers and ganglion cells, and thickening of the vessel walls, so that in some instances their lumen was obliterated (obliterating arteritis).

Symptoms.—These consist of paroxysms of sharp, shooting, burning pain in the course of the affected nerve. In the intervals between the paroxysms there may be constant dull pain. The pain is increased or brought about by irritation, as by cold, heat, or pressure. There is frequently hyperesthesia in the region of the affected nerves; but firm, long-continued pressure may give relief. In some cases tender points may be found which correspond to the exit

[1] Amer. Jour. Med. Sci., 1898, p. 503.

of the nerve from a bone canal or from the muscle or fascia (points of Valleix). Vasomotor, secretory, and trophic disturbances may occur. Frequently the paroxysms may recur at the same time each day. Now and then we meet with a patient in whom a painful impression remains after the true paroxysm has passed away; this is called "reminiscent" or "hallucinatory" neuralgia.

Diagnosis.—We have already spoken of the features which distinguish neuralgia from headache (p. 104). The diagnosis from neuritis would depend upon the facts that in *neuritis* the pain is constant, not darting and shifting; there is tenderness along the whole course of the nerve, made worse by firm pressure; there is frequently anesthesia, and motor paralysis occurs.

The various *forms* of neuralgia will be discussed under diseases of the "cranial and spinal nerves," when treatment will also be discussed.

PARESTHESIA

Definition.—The general term "paresthesia" has already been defined (p. 47). These sensations sometimes confine themselves to a certain locality or occur in the distribution of a certain nerve, and may be then considered as a disease. When the feet and hands separately or together are affected we call the disorder *acroparesthesia;* when it only occurs upon arising in the morning, we speak of *waking numbness;* and when confined to the distribution of a particular nerve, as the ulnar or external cutaneous, *meralgia paræsthetica* is the term used.

Etiology.—Paresthesia may occur in either sex, usually after middle life; but is more common in women, being especially frequent in women during the menopause and in those who do scrubbing, washing, and sewing. Those of either sex who are obliged to be upon their feet a great deal are prone to it. The gouty or rheumatic diathesis and alcoholism are causes. It has followed the infectious diseases.

Pathology.—Paresthesia is probably due either to a poison circulating in the blood, which irritates the sensory nerve

endings, or to a disarrangement of the blood supply of the nerves, either hyperemia or anemia. In some cases there is most likely a low grade of neuritis.

Symptoms.—These consist of feelings of numbness, tingling, or coldness, usually beginning in the hands, and as time goes by the arms, feet, and legs becoming involved. These symptoms are often felt just after awaking in the morning, and pass off as the limbs are used (waking numbness); but if the disorder lasts long enough, the sensations become constant. They may be so severe as to awaken the patient. The scalp and ears may rarely be affected. There is usually no muscular weakness or anesthesia, but a clumsy feeling may be present in the fingers, so that fine movements are performed with difficulty. Cases have been described by Roth, Osler, and others in which paresthetic sensation, and sometimes pain, was felt in the region supplied by the external cutaneous nerve on the outer side and front of the thigh (meralgia paræsthetica). This form is aggravated by standing. It has been caused by the pressure of a corset. Similar symptoms may also occur in the anterior surface of the thigh, the region supplied by the middle cutaneous nerve (meralgia paræsthetica anterior).

In *waking numbness* there is sometimes temporary weakness. General restlessness and nervousness are usually present. The urine may be excessive in amount and contain large amounts of urates or phosphates. Vasomotor symptoms, characterized by local congestions and sweating, are sometimes seen.

Diagnosis.—Acroparesthesia must be distinguished from neuritis, organic disease of the brain and cord, hysteria, neurasthenia, and Raynaud's disease. There is no tenderness of the nerve trunks, muscular paralysis and atrophy, or anesthesia, which we find in neuritis. Paresthesia may be a symptom of organic disease of the brain, cord (especially tabes dorsalis), and peripheral nerves; of neurasthenia and hysteria; but when present in any of these conditions, other symptoms characteristic of the disorder would be found, and the paresthesia would not be limited in distribution and constant in duration as in the forms we have

described, but diffuse and temporary. Raynaud's disease is characterized by extreme pallor of the affected parts, followed by congestion, symptoms which are not present in acroparesthesia (p. 494). The latter, however, has been supposed by some to be an abortive form of the former.

Prognosis.—The disease is obstinate to treatment, and relapses are apt to occur; it does, however, often get well. It never progresses to any serious disorder.

Treatment.—Ergot in large doses, long continued, as recommended by Sinkler, often does good. Faradization, galvanization, the anode being applied labile, and the high frequency current applied with a vacuum electrode have been of service. Massage of the affected limbs is also useful. In those cases in which gouty or rheumatic diathesis is suspected, salicylates, lithia, alkalies, and a proper diet should be advised. Tonics, as strychnine, arsenic, mineral acids, and iron, must be given after the symptoms subside; and a change of air and rest, if practicable, be secured. In obstinate cases of meralgia paresthetica, stretching or resection of the external cutaneous nerve has been suggested by Spiller.

VERTIGO

Definition.—Vertigo, familiarly spoken of as giddiness or dizziness, is the consciousness of disturbed equilibrium due to a derangement of the nervous mechanism which governs the relations of the body to external objects.

Etiology.—The causes of vertigo are: (1) Visual defects, as refractive errors and loss of muscle balance. (2) Aural disturbances, as either disease of the labyrinth (Ménière's disease), impacted cerumen, or inflammatory conditions of the middle ear, causing irritation of the auditory nerve endings in the semicircular canals. (3) Toxemic conditions, as at the onset of infectious diseases, alcohol, tobacco, lithemia, dyspepsia, constipation. (4) Causes which produce derangements of the cerebral circulation, as arteriosclerosis, hyperemia, anemia, valvular disease of the heart, and fatty heart. (5) Neuropathic conditions, as neurasthenia, hysteria. (6) Organic disease of the brain, especially

of the cerebellum and its peduncles. (7) Mechanical causes which produce disarrangement of the fluid in the semicircular canals, as swinging, sea voyages (sea-sickness), rotary movements, etc.

In a few cases no cause can be detected; these are known as "essential vertigo."

Pathology.—For the securing of the balance or equilibrium of the body in its changing relations to external objects, accurately timed and ever-changing muscular contractions are necessary. The motor impulses necessary to cause these contractions are determined in the cortical centres by certain sensory impressions which are constantly being received for their guidance. These impressions are derived from the eye and its muscles; the semicircular canals of the internal ear; from the skin of those parts in contact with external objects; and from the articular surfaces and muscles about them. These impressions are correlated in the cerebellum with which the vestibular nerve is connected. This nerve has connections with the ocular nerves and pneumogastric. Sensory impressions from the joints and muscles also pass to the cerebellum. Any cause which disturbs either the flow or reception of sensory impulses from these sources causes a derangement in time and force of the motor impulses. This is manifested by vertigo. The nausea, vomiting, and irregular pulse are due to reflex irritation of the vagus nerve.

General Symptoms.—An attack of vertigo comes on suddenly, and as a rule is of momentary duration. Objects seem to whirl about the patient; the floor or bed rises or sinks (objective vertigo); or the patient himself feels as if he were whirling rapidly about (subjective vertigo). There is apt to be mental confusion, but rarely loss of consciousness; the patient totters and sometimes falls; there may be nausea and vomiting, and irregular pulse often occurs in severe attacks. In attacks due to toxic substances, as alcohol, tobacco, and other narcotics, the vertigo is apt to last a considerable time. Sudden rising or other movements may increase the vertigo. Attacks may sometimes be produced when the head is held in certain positions. *Auditory* vertigo deserves special mention.

Auditory Vertigo.—*Inflammatory conditions* of the middle ear or impacted cerumen in the canal may produce vertigo. They possibly do this by causing irritation of the auditory nerve endings, and in some instances by the pressure of the stapes upon the labyrinthine fluid. These forms must not be confounded with

Ménière's disease, which is due to primary disease of the internal ear involving the end organs of the eighth nerve in the labyrinth. The lesion in *chronic cases* may be caused by exposure to cold, gout, or syphilis; or to atrophy of the auditory nerve. Some of the *acute cases* are due to hemorrhage.

The affection is rare in early life. The patient suffers from deafness of nerve origin (p. 58), tinnitus, and paroxysms, occurring at regular intervals, of intense vertigo. The erect posture is impossible, and the recumbent position even does not relieve it. The paroxysm is usually terminated by nausea or vomiting, and sometimes syncope. The paroxysms may occur daily, or weeks may intervene.

The acute or apoplectiform attacks begin suddenly with tinnitus and paroxysms of vertigo as just described. In the intervals there is apt to be impairment of equilibrium, so that the patient walks with difficulty. The prognosis is uncertain. A small proportion of cases may recover with total loss of hearing on the affected side. In the majority of instances the disease, with intervals of improvement, continues through life.

It is important to remember that tumors of the cerebellopontile angle may cause similar symptoms (p. 356).

Diagnosis.—The presence of the symptom may usually be recognized without difficulty. In some instances the possibility of the attack being due to epilepsy in the form of petit mal must be considered. In this, however, loss of consciousness can always be discovered; the attack is of shorter duration; and giddiness, nausea, and vomiting are not present as in vertigo. The cause must always be determined if possible. This is done by a careful physical examination of the heart, eyes, ears, bloodvessels, etc., of the patient, and inquiry into the associated symptoms. The possibility of epilepsy must be borne in mind.

Prognosis.—The prognosis depends upon the cause. If that can be corrected, it is good. In that due to organic brain disease or Ménière's disease it is of course not favorable.

Treatment.—The treatment of vertigo consists of (1) that of the attack; (2) that of the cause.

The *attack* is best treated by rest in the recumbent position, and the administration of aromatic spirits of ammonia and the bromides. The causes are mentioned on p. 112.

In *Ménière's disease* quinine in ascending doses until cinchonism results, when the drug is stopped for a time (Charcot). Or the use in moderate doses of the salicylates may be tried. Sinkler recommends the long-continued use of ergot and cannabis indica. The continuous use of the bromides and the hypodermic injection once daily (Hirt) of ten drops of a 2 per cent. solution of pilocarpin have been of service. In desperate cases the stapes has been removed. Removal of the semicircular canals has also been done,[1] and Ballance[2] recommends cutting the auditory nerve. These measures while relieving the symptoms cause deafness.

Vertigo due either to arteriosclerosis or senility is benefited by nitroglycerin and potassium iodide. The treatment of the other forms consists of the removal of the cause, when possible.

DISORDERS OF SLEEP

Insomnia.—Definition.—Insomnia is the term used to designate a condition characterized by habitual incomplete sleep, or by periods of entire absence of normal sleep.

Etiology.—The physiology of sleep is not well understood. It has been found that during sleep there is anemia of the brain accompanied by dilatation of the bloodvessels of the limbs; but as anemia from disease does not cause an excessive desire for sleep, it is evident that this is not the cause. Wilcox advances the view of Rabl-Ruckhard that sleep is due to a retraction of the dendritic processes of the neurone,

[1] Review Neurol. and Psychiat., November, 1908, p. 646.
[2] Ibid., December, 1908, p. 730.

which, of course, would prevent for the time being communication between the different parts of the nervous system and interchange of nerve impulses.

Symptoms.—Insomnia may be a symptom of any of the organic and functional diseases of the nervous system. It is especially common in the neurasthenic and overworked. It is also a symptom of disordered action of other organs, viz., in toxic states of the blood from infectious disease, kidney diseases, lithemia, or drugs, as tea, coffee, alcohol, and in diseases of the heart and bloodvessels.

Its prolonged presence causes mental depression and irritability.

Treatment.—Treatment should, of course, be directed to the cause of the symptom when it can be ascertained. In treating insomnia drugs should be avoided when possible.

Often one of the following measures, used before retiring, will answer: muscular exercise, massage, a cup of hot milk or bouillon, or a hot bath (104°), continued until the cutaneous surface is reddened. A season of camp life may bring relief when all else fails.

When drugs are required a thirty-grain dose of bromide of sodium will often suffice. In the insomnia of neurasthenia, one of the bromide salts, as ammonium or strontium, in ten-grain doses t. i. d may prove useful. If these are not successful either trional, gr. xv–xxx in hot milk, an hour or two before bedtime, or veronal in doses of gr. v–viij given the same way are useful. Chloral, gr. xv–xx, or by the method of Ringer, gr. v every half-hour until four or five doses are taken, is of much service. Sulphonal and paraldehyde are also of use. When accompanied by excitement or agitation hypodermic injections of hydrobromate of hyoscine, gr. $\frac{1}{100}$, repeated every four hours as required, is most useful. Opium should never be prescribed for insomnia unless caused by pain. To avoid the formation of *drug habits*, whatever drug is used it is well, if possible, to prevent the patient from either knowing what it is or being able to renew the prescription.

Morbid Drowsiness.—This may occur in two forms: either a continuous drowsiness, or sleep extending over

hours or days, or paroxysmal attacks of an imperative desire for sleep which only lasts a few minutes. To the latter attacks the term *narcolepsy* has been applied.

As causes for the former attacks, old age; vascular degeneration; various forms of toxemia, especially uremic, diabetic, and syphilitic; cerebral tumor, exhausting diseases, and hysteria may be mentioned. It may also follow the status epilepticus. In most of these cases the condition resembles stupor more than true sleep.

In the latter attacks the sleep, which only lasts a few minutes, resembles natural sleep and may be accompanied by dreams. When the desire occurs, it is imperative, the patient going to sleep no matter what he may be doing. It is a rare neurosis. Care must be taken not to confound these attacks with petit mal (p. 438).

Treatment.—The treatment of the prolonged attack is of the cause. In some cases of narcolepsy caffeine has been of service, in others static electricity.[1]

[1] See McCarthy, Amer. Jour. Med. Sci., 1900, p. 178; also Camp, Jour. of Abnormal Psychology, April-May, 1907.

CHAPTER VI

DISEASES OF THE PERIPHERAL NERVES

COMPRESSION PALSY OR PRESSURE PALSY

Definition.—Pressure palsy is a paralysis of certain muscles due to long-continued pressure upon the nerve supplying them.

Pathology.—The compression, in producing these symptoms, acts by separating the molecular elements of the white substance, and setting up a secondary degeneration of the same character as results from the division of a nerve. (Gowers.)

Symptoms.—When a nerve trunk is subjected to slight pressure, if long continued, numbness, tingling, and a feeling of heaviness, and at times transient inability to move the limb, are experienced. These sensations are familiar to all, and are commonly described by saying that the part is "asleep." When the pressure is more pronounced or of longer duration the symptoms do not pass away, and a paralysis of the muscles supplied by the compressed nerve results. The musculospiral, from the frequent habit of sleeping with the head resting upon the arm, is especially liable to receive pressure, and is the nerve most commonly affected. A history of previous indulgence in alcohol is frequent in these cases. Other nerve trunks that are more or less commonly affected are the ulnar, sciatic, and anterior tibial. The patient complains of a feeling of numbness and inability to use the muscles. There is no tenderness over the nerve trunk, and usually no anesthesia. Atrophy takes place, but it is not excessive, as a rule. The electrical reactions are either normal or in aggravated cases there is a quantitative decrease to galvanism and faradism.

Diagnosis.—The diagnosis of pressure palsy is based upon the discovery that pressure, as from crutches, sleeping with the head upon the arm, etc., has been exerted upon the nerve. The absence of pain and tenderness distinguishes the condition from neuritis. When the musculospiral nerve is the seat of the trouble neuritis due to lead might be thought of; but the absence of other symptoms of lead poisoning and the fact that the paralysis is usually unilateral, distinguish it from that.

Treatment.—Measures to promote the nutrition of the muscles, as electricity, massage, and strychnine, either hypodermically or by the mouth, are indicated.

NEURITIS OR INFLAMMATION OF NERVES

Neuritis may be confined to a single nerve trunk and be *local;* or a number of nerves may be affected, when it is termed *multiple.* It may be interstitial or parenchymatous. It may be either acute or chronic. The acute form often has a tendency to subside into the chronic.

Morbid Anatomy.—Inflammation of a nerve may be *interstitial,* in which the connective tissue is the primary seat of the process; or *parenchymatous,* when the nerve fibers are diseased without involvement of the connective tissue.

In *acute interstitial neuritis* the changes occur in the perineurium and endoneurium. The nerve is soft, swollen, and reddish in color. Microscopically we find the blood-vessels distended; infiltration of round cells between the nerve bundles; and the perineurium and connective-tissue trabeculæ are increased in size. In very advanced forms partially degenerated fibers, with fatty myelin sheaths and swollen axis cylinders, are found; ultimately the nerve fibers may be destroyed. The changes in the nerve fibers are secondary to those in the connective tissue.

Chronic interstitial neuritis may follow the acute form or occur independently. The nerve is hard, the connective tissue increased; the bloodvessel walls are thickened, their lumen sometimes obliterated; and more or less round-cell

infiltration is found in the connective tissue. The nerve fibers are degenerated, and many of them have disappeared. Frequently there is proliferation of the cells in the neurilemma. Ascending degeneration in the posterior columns of the cord and changes in the ganglion cells may be found.

In *parenchymatous neuritis* we find the nerve trunks slightly firmer and grayer than normal. The usual evidences of inflammation are absent; the myelin is segmented and divided into drops and granules; and the axis cylinders are granular, subdivided, and finally disappear. The nuclei of the sheath of Schwann proliferate, and finally the nerve becomes a fibrous cord. Changes are sometimes found in the anterior ganglion cells in the cord (reaction at a distance).

Localized Neuritis.—Etiology.—Localized neuritis arises from: (1) Exposure to cold. (2) Traumatism, as wounds; blows upon the nerve; the tearing and stretching which follow a dislocation or fracture; and electrical shock. (3) The extension of inflammation from neighboring parts, as neuritis of the facial nerve due to inflammation of the middle ear; inflammation of joints; septic wounds of the extremities, etc. (4) Arteriosclerosis, arteritis obliterans. (5) Poisons, as the infectious diseases, alcohol, and metallic poisons. These, however, usually cause multiple neuritis (*vide*). (6) Stoop shoulders, which cause compression of the axillary structures between the humerus and the ribs, and hence either brachial neuritis or ulnar neuritis, may result.[1]

Localized neuritis is usually interstitial.

Symptoms.—Slight constitutional disturbances may be present. A most prominent symptom is pain in the course of the nerve and parts to which it is distributed. This is of a burning, boring character, worse at nights and increased by movements of the affected part. The nerve is tender when pressed upon. Occasionally redness of the skin over it and edema are present. The function of the muscles supplied by the affected nerve is impaired, and they become flaccid and finally atrophy. Changes in their response to

[1] Goldthwaite, Jour. Amer. Med. Assoc., September 11, 1909, p. 852.

the electrical currents, varying from a quantitative decrease in mild cases to a typical De R (p. 77), are found. At the onset muscular twitchings are sometimes noticed. Various paresthetic sensations are experienced. Hyperesthesia may be present, and after a time sensation may be lessened, or total anesthesia in small areas is found. The skin becomes atrophied and glossy, or more rarely thickened, and the nails are ridged and brittle. Increased perspiration has been observed. These symptoms vary in severity according to the acuteness of the process.

Constitutional symptoms are absent in subacute and chronic cases, the pain and tenderness usually are not so severe, and in the latter, when it does not follow an acute attack, the development of the symptoms is gradual. In these, contractures due to overaction of sound muscles may develop. When the inflammation is confined to the connective tissue and there is not much exudation the function of the nerve fibers is not much impaired. In these cases pain and tenderness may be the only symptoms noticeable, but careful examination will also show some flaccidity and weakness of the muscles. The inflammation, when due to septic processes, may extend up the nerve trunk and involve other nerve trunks in communication with it. It may even reach the spinal cord. This is known as *ascending* or migratory neuritis. It is rare.

The **duration** of a neuritis may vary from a few weeks in mild cases to months and even years.

Diagnosis.—From the ordinary rheumatic attack localized neuritis is differentiated by the localization of the pain and tenderness to the course of a nerve, and the impairment of function of the muscles supplied by this nerve. In this connection it must be remembered that in very acute attacks the pain may be diffuse and the muscles tender; but investigation of the nerve trunks and the quickly resulting muscular disability and atrophy will settle the question.

Chronic neuritis, especially those mild cases in which the motor symptoms are not marked, may be mistaken for neuralgia; but in *neuritis* pain and tenderness of the nerve are constant; in *neuralgia* the pain is darting and

paroxysmal, and tenderness, when present at all, is so only during paroxysms and is confined to spots of definite localization (tender points of Valleix). In neuritis there is usually some degree of weakness and flaccidity of the muscles; in neuralgia there is none.

The pains due to some cord diseases may simulate neuritis; but there are not local tenderness of the nerve and limitation of function in the distribution to a single nerve.

Volkman's contracture or *ischemic paralysis* may be mistaken; this condition is always produced by the pressure of splints on the forearm, the flexor muscles are affected, sensation is not disturbed, and any muscle tissue left reacts normally to the electric current.

Prognosis.—This is always doubtful. Apparently mild cases are often intractable to treatment. The gravest forms are those due to secondary infection from a local suppurative inflammation. As the symptoms are more intense, especially the evidences of neural degeneration as seen by muscular atrophy and well-marked De R, the prognosis becomes worse. In neuropathic subjects the pain is apt to remain even after all other symptoms have vanished. Ascending neuritis is always most obstinate.

Treatment.—In acute neuritis rest of the part where the inflamed nerve is situated is of the first importance. For the relief of pain, antipyrin, phenacetin, and like drugs can be administered and the part wrapped in *hot* lead-water and laudanum or other hot applications. Care must be taken not to blister the skin with these, as troublesome sores may remain. If the patient is gouty or rheumatic, salicylates in full doses should be administered. In the author's experience one of the most efficient means for the relief of pain is the passage of a constant galvanic current down the course of the nerve for ten or fifteen minutes daily. In some cases this may aggravate the pain, when it should be discontinued. Ointments containing ichthyol and belladonna, and a 25 per cent. ointment of mesotan, are also useful.

After the more acute symptoms have subsided, vigorous counterirritation by means of fly-blisters or the cautery should be substituted for the lead-water and laudanum. In

this stage the use of the extreme dry heat (250° to 300°), applied by an apparatus devised for that purpose, is useful.

When the acute stage is past or has subsided into the chronic, in conjunction with counterirritation, measures to promote the nutrition of the muscles—viz., massage, electricity, and strychnine, preferably hypodermically—must be employed. In ascending neuritis vigorous counterirritation should be maintained above the area of inflammation; and the question of resection of the nerve or even amputation above the seat of inflammation may sometimes have to be considered.

Primary Brachial Neuritis.—Definition and Etiology.—The term is used to designate a primary inflammation of several or all of the nerves which enter and make up the brachial plexus (Herter).

It is a rare disease, which occurs during the second half of life (after fifty), and is usually met with in debilitated and dyspeptic people, who sometimes in addition have a history of gout or rheumatism. They may previously have suffered from neuritis elsewhere, most frequently in the sciatic nerve.

The **pathological process** is usually confined to the nerve sheaths (perineuritis), and in some cases is principally or altogether confined to the nerve roots which make up the plexus. This is termed radicular neuritis.

Symptoms.—The prominent feature of these cases is pain, which is most intense. This may begin at a distance from the plexus, back of the forearm, the wrist, and scapular region; but soon becomes most intense in the axilla and supraclavicular region, and radiates down the arm in the course of the nerve trunks. The pain is dull, aching, with sharp, shooting exacerbations, and is aggravated by movement. Hyperalgesia of the skin over the plexus is usually present. Less frequently slight degrees of anesthesia are found. Loss of power is not excessive. Atrophy, changes in the electrical reactions, trophic and vasomotor disturbances, as thinned and glossy skin, local edema, and in old cases adhesions in and swelling of the joints, are symptoms.

Diagnosis.—The disease may be confounded with neuralgia, but the persistent tenderness, muscular weakness, and

atrophy present in neuritis are not symptoms of neuralgia. Similar pain in the arm might be produced by aortic aneurysm; but tenderness would not be found, and careful examination would cause discovery of the aneurysm. The presence of a cervical rib may also produce similar symptoms, but a skiagraph will reveal its presence.[1] The late joint changes resemble those of rheumatoid arthritis, but the history of previous pain and tenderness in the course of the nerves would distinguish the neuritis. The symptoms of radicular neuritis may be confounded with those of vertebral disease, of meningitis, or tumor in the cervical region. In these, however, there would not be nerve tenderness; while in bone disease there would be tenderness on pressure and jarring, and muscular rigidity; and in meningitis or tumor there would also be evidences of pressure upon the cord.

Prognosis.—The affection is usually of long duration, a year or more. The pain may continue after evidences of neuritis have disappeared. Owing to the changes in the joints, permanent disability and deformity may result. Relapses may occur.

Treatment.—Absolute rest, by placing the arm upon a splint or bandaging it to the side, must be enjoined. Cocaine, hypodermically, beginning with $\frac{1}{10}$ grain and increasing, will usually relieve the pain, but must be used cautiously for fear of provoking the habit (Herter). The general health of the patient must be built up. Otherwise the treatment is that of neuritis in general (p. 122).

Neuritis of some special nerves may demand some modification or addition to this treatment, which will be described when we describe the diseases of these nerves.

Multiple Neuritis.—Etiology.—Multiple neuritis is probably always due to the influence of a poison circulating in the blood; although in some cases we may be unable to determine its nature. These causes are expressed in the following table, modified from that of Ross:

I. *Idiopathic form* (due to an unknown poison): (*a*) Acute; (*b*) subacute; (*c*) chronic; (*d*) recurrent.

[1] C. M. Hinds-Howell, Lancet, June 22, 1907.

II. *Poisons introduced from without:* (*a*) *Diffusible stimulants:* alcohol; carbon monoxide; carbon bisulphide; dinitrobenzine; aniline. (*b*) *Infectious diseases:* diphtheria; influenza; typhoid fever; scarlatina; measles; pertussis, etc.; septicemia; syphilis; tuberculosis; pneumonia; malaria; chorea; beriberi; leprosy. (*c*) *Metallic poisons:* lead, arsenic, mercury; phosphorus; silver; cyanide of potassium.[1] (*d*) Ptomains.[2]

III. *Poisons produced within the body:* Gout; rheumatism; pregnancy and the puerperal state; diabetes.

IV. *The dyscrasic form:* Chlorosis; marasmus; cancer and other forms of cachexia; vascular degeneration.

Multiple neuritis may occur at any age.

Morbid Anatomy.—The inflammation in these cases is almost invariably of the parenchymatous variety (p. 120).

Symptoms.—All of the causes above mentioned do not produce similar symptoms. From some, sensory nerve fibers especially suffer; from some, the motor; while from others both motor and sensory fibers are alike affected. Again, certain poisons seem to have a predilection for certain nerve trunks; for instance, that of lead for the musculospirals For these reasons we have certain types due to certain causes, these types being sufficiently constant to enable us in many cases to be almost positive of the cause from the symptoms produced.

Some of the causes mentioned are but rarely the cause of neuritis, both because the individual is infrequently brought under the influence of many of them, and because the nerves seem to be more susceptible to the toxic influence of some than of others. Only the more common forms will be here described.

Idiopathic Multiple Neuritis.—This may occur at all ages from infancy to old age, the majority of the cases occurring during middle life. In some cases it appears to follow chilling of the body after being overheated or exposure to cold. In most of them no cause can be ascertained. The

[1] Jour. Nerv. and Ment. Dis., July, 1908, p. 417.

[2] Spiller has reported a case of multiple neuritis due to the ingestion of putrefying pork. Philadelphia Polyclinic, vol. vii, p. 455.

attack usually begins with a chill followed by a rise of temperature, pain in the back and limbs, headache, and general malaise. Paresthetic sensations are felt in the hands and feet; the skin may be hyperesthetic and the muscles and nerves are tender.

Loss of power soon becomes manifest. It usually begins in the muscles supplied by the external popliteal nerve and extends upward; more rarely the arms may be first involved. The paralysis extends until in severe cases the muscles of the trunk and those of respiration are involved. Atrophy of the muscles occurs early; the deep reflexes are lost; those of the skin may be present or not. In the early stages there is merely a more or less quantitative decrease in the response of the muscles to the faradic current. As the disease progresses partial reactions of degeneration may be found. There may or may not be areas of anesthesia present.

Recurrent Multiple Neuritis.—In 1891 Dr. Mary Sherwood reported two cases in which, without apparent cause, the patients had had several attacks of neuritis. Since then a few others have been reported, notably by H. M. Thomas, whose case was then in the fifth attack of multiple neuritis. The symptoms of these cases are the ordinary ones. It has been noted that rarely patients who have had lead neuritis have recurrences without renewed exposure. No evidences of lead, however, were present in most of the cases reported as recurrent neuritis, and the cause of these attacks remains doubtful.

Alcoholic Multiple Neuritis.—Alcohol is a frequent cause of neuritis. It is more apt to occur in persons who drink steadily than those who indulge in periodical sprees. Women appear to be especially liable.

The first symptoms are sensory disturbances, pain in the extremities, with pins-and-needles sensations, and a feeling of numbness. There is marked hyperesthesia of the calf muscles. Muscular weakness soon appears, the extensors of the feet being usually first affected, those of the hands soon following. Double foot-drop and wrist-drop are then produced. The former is a most characteristic symptom of the disorder (Fig. 33). Eventually the other muscles of the

limbs become paralyzed. The motor cranial nerves may become affected, and rarely the optic. The sphincters are not usually involved. Marked muscular atrophy soon ensues, and changed electrical reactions, from quantitative decrease to reactions of degeneration of varying degrees, are present. The knee-jerk is lost early;[1] the skin reflexes are usually active. Severe pain is always present; the nerve trunks and muscles are exceedingly tender, the latter more so than in any other form of neuritis, and the skin is hyperesthetic. Trophic symptoms are rare, but glossy skin and roughening of the nails may occur. Excessive sweating is sometimes seen. In severe cases there is elevation of

FIG. 33

Multiple neuritis. Double wrist-drop and double foot-drop. (Lloyd.)

temperature, the pulse is rapid and weak, and the capillary circulation bad. Mental symptoms, are often present. In most cases these do not resemble delirium tremens, but it should be remembered that it may coexist. (See Korsakow's Psychosis, p. 534.)

When the patient becomes able to walk the gait is characteristic; owing to the foot-drop the knees must be lifted high in order to clear obstacles—the so-called "steppage" gait. Incoördination may then be also a feature, and the disease is then sometimes known as pseudotabes.

[1] In some cases it may be present during the early or irritative stage.

Multiple Neuritis from Lead.—This form is peculiar in that the posterio-interosseous branch of the musculospiral nerves is usually the first to be affected, causing paralysis of the extensors of the wrist and fingers (p. 189). For some unknown reason the supinator longus and the extensor ossis metacarpi pollicis, also supplied by the musculospiral nerve, escape. One arm, usually the right, may be affected first; but the other soon follows, and the patient in a short time has a double wrist-drop, atrophy of the affected muscles rapidly occurs, and the reaction of degeneration in various degrees is present. The supinator longus, not being diseased, stands out prominently. There is usually neither pain nor tenderness. Rarely slight tenderness of the nerve trunks and spots of anesthesia may be found. The disease may remain limited to the musculospirals, or may spread to other nerves, causing paralysis of other muscles of the arm, especially the biceps and deltoid. The leg may be affected. The deep reflexes are absent.

There is another type, not so common as the above, in which atrophy is first noticed in the adductor muscles of the thumb and intrinsic muscles of the hand (lumbricals and interossei), and the symptoms resemble in their progress those of progressive muscular atrophy (p. 269). In these cases there is involvement of the ganglion cells in the anterior horns of the cord.

Arsenical Neuritis.—It is important to remember that arsenic given in medicinal doses has caused neuritis. The symptoms consist of paralysis of the extensors of the arms and legs; muscular atrophy; pains of a shooting, darting character; tenderness over the nerve trunks, and frequently anesthesia. Eruptions and pigmentation of the skin are often present.

Diphtheritic Neuritis.—The paralysis usually makes its appearance during convalescence, but may occur in the midst of the attack. It begins in the muscles of the palate, evidenced by a nasal voice and difficulty in deglutition and regurgitation of liquids through the nose. Inspection shows the palate to hang lower than usual, and that it is not elevated when the patient utters the sound "ah." In mild cases the

paralysis may remain confined to this part. More usually it extends to muscles of the eyes, especially the ciliary, evidenced by impaired vision and loss of the power of accommodation. The arms and legs may then become affected. In 125 cases analyzed by Goodall,[1] paralysis of the palate was noticed first in 83; of the ciliary muscles in 20; of the palate and ciliary muscles in 5; in 4, of the palate and lower extremities; in 3, of the lower extremities; in 2, of the muscles of the pharynx; and of the muscles of respiration in 2. In the remaining 6 cases the parts first affected were scattered, one case each, over various other parts of the body.

When the limbs are affected the muscles soon become flaccid and atrophy, and the reaction of degeneration in various degrees is found. The knee-jerk is absent—in fact it may be absent before other signs of paralysis are manifest. Sensory disturbances are most frequently absent. If present, they consist of numbness, tinglings, and anesthesia.

The heart's action is often rapid and irregular, due to involvement of the pneumogastrics. In rare instances there are incontinence of urine, and in adults loss of sexual power.

Beriberi, endemic neuritis, or **kak-ke,** is probably due to a specific microörganism. Diet has been thought to have considerable influence in its production. Vorderman, a Dutch physician, has stated that those who eat white rice, that which has been deprived of the pericarp, are much more liable to suffer than those who eat the red, from which it has not been removed. It is met with in Japan, China, Philippine Islands, Ceylon, parts of India, and other tropical localities. It also occurs among sailors on ships carrying cargoes of sugar. Intestinal parasites have been found to be very prevalent in those suffering from beriberi. Lately it has been denied that the diet has very direct influence in its causation, but that it is an acute, infectious disease.

Recent investigations seem to show that to a certain extent

[1] E. W. Goodall, Brain, 1895, p. 282.

it may be spread by direct contagion.[1] Lack of nutrition, bad food, and exposure to cold and damp act as predisposing causes. The germ enters the body through the alimentary canal and multiplies in the duodenum.

Symptoms.—The symptoms are the usual ones of multiple neuritis, viz., pain, tenderness, muscular weakness and atrophy, anesthesia in varying degrees, associated with edema, a tendency to effusion into serous cavities, cardiac disturbance, and general malaise. In some cases the edema and serous effusion are absent, and these cases are attended with extensive paralysis, even of the facial muscles, and excessive pain. In others, the pain, tenderness, and atrophy are absent, the symptoms consisting of edema, weakness, and loss of knee-jerk (moist variety).

The pneumogastric, phrenic, and cardiac plexuses of nerves are apt to be affected, when death ensues. Degeneration of the peripheral nerves is found in the ordinary cases, while in the moist cases the phrenic, sympathetic, and vasomotor nerves only are involved.

Senile neuritis occurs in old age and is probably due to arteriosclerosis. The symptoms develop slowly and consist of weakness of the limbs, usually the feet and legs, and numbness. Sometimes there is slight atrophy and diminished response to electricity. The knee-jerks are lost. Cranial nerves may also be affected either in connection with spinal ones or alone. Tenderness is absent. Evidences of vascular disease will be found and other causes of neuritis will be absent. The treatment consists in the administration of nitroglycerin, caffeine, strophanthus, and other measures used in the treatment of arteriosclerosis.

Other Forms of Multiple Neuritis.—The remaining forms resemble, in their general features, those just described.

Diagnosis.—There should not be any difficulty in making diagnosis of multiple neuritis. The diseases most likely to be confounded with it are acute poliomyelitis, acute ascending myelitis, and locomotor ataxia. The differences between it and poliomyelitis are well shown in the following modification of the summary by Starr:

[1] Brain, 1903, p. 488.

Acute poliomyelitis.	Multiple neuritis.
Sudden onset, with fever and development of paralysis in all the limbs, followed in from three to five days by subsidence of paralysis, which remains in a few muscles of one limb; or, if two are affected, the paralysis is rarely symmetrical. If the onset is subacute, four weeks are the duration of onset. The muscles are not tender. Sensory symptoms are rare, and when present soon subside.	Fatigue for some weeks; then sudden onset and progress for two weeks, with or without fever. Legs usually first affected, then arms, then body. The paralysis has no tendency to subside for some time (months). The limbs are affected symmetrically. Usually there are tenderness of the nerve trunks and affected muscles, and pain, with areas of anesthesia.

In *acute myelitis* the loss of sensation is complete, and begins at a well-marked line which extends around the body at the level of the cord lesion and involves all parts below it. A girdle pain is usually present. There is incontinence of urine and feces, and bed-sores develop. There is no tenderness of the nerve trunks.

In *locomotor ataxia* there is absence of muscular and nerve tenderness, muscular weakness and atrophy, and the Argyll-Robertson pupil can usually be found.

Idiopathic multiple neuritis may be mistaken for *Landry's paralysis;* in this, however, there are but slight, if any, sensory symptoms, and no changes in the electrical reactions.

The fact that the neuritis is due to alcohol can be determined by the history of steady indulgence, although in women this may be often denied; by the intense pain and muscular tenderness, especially of the calves; by the peculiar distribution of the paralysis (extensors of both arms and legs); and by the frequent accompanying mental symptoms.

That *lead* is the cause can be recognized by finding the blue line upon the gums, often a history of preceding lead colic, and exposure to the poison; the absence of pain and tenderness; and the paralysis first appearing in and often confined to the muscle supplied by the musculospirals, excepting the supinator longus and extensor ossis metacarpi pollicis.

In musculospiral paralysis from other causes these muscles do not escape, and the trouble will be most likely unilateral.

Diphtheritic neuritis is peculiar in the frequent absence of sensory symptoms, the muscles of the throat and eye being

first attacked. When the muscles of the extremities are primarily paralyzed an attack of poliomyelitis might be suspected; but the previous history of recent diphtheria, the symmetry and frequently progressive character of the paralysis, and slighter changes in the electrical reactions which distinguish the neuritis, are not characteristic of poliomyelitis (p. 257). It must be remembered also that there are other forms of palsy associated with diphtheria. As in other acute infectious diseases, sudden hemiplegia due to either thrombosis, embolism, or hemorrhage may occur. This form resembles cerebral hemiplegia from other causes (p. 322). Diphtheritic paralysis may be accompanied by a rapid and complete palsy of one or more of the cranial nerves, which is apt to be permanent, and is probably due to an acute inflammation of their nuclei (encephalitis).

In cases of multiple neuritis which do not conform to the types above described, careful investigation must be made for the existence of any of the rarer causes mentioned.

Prognosis.—In patients previously healthy and in whom the heart or respiratory muscles do not become paralyzed, recovery is the rule. Before this is complete, months will usually elapse. Some muscles may never entirely recover, and then contractures are apt to result. Sensory symptoms always disappear first. Idiopathic, alcoholic, and diphtheritic neuritis are especially liable to a fatal issue, owing to the liability to involvement of the nerves supplying the heart and respiratory muscles.

Treatment.—When possible the cause should be removed. In alcoholics this must be done with caution, large doses of strychnine and other heart tonics being substituted. Absolute rest in bed is essential, excepting in those cases due to lead, in which the arms alone are affected. For the relief of pain, similar measures to those recommended for local neuritis may be employed. Owing to the depressing effects of the drugs recommended for this purpose, salicylates and others, they must be used cautiously, and strychnine or caffeine are advantageously combined with them. Morphine should only be used as a last resort. After the acute symptoms have subsided, measures to increase the nutrition of

the muscles are indicated, as massage; causing muscular contractions by electricity; and tonics, such as strychnine in large doses, especially indicated in the alcoholic form. Cod-liver oil, iron, and small doses of bichloride of mercury or iodide of potassium may also be useful in this stage. The food during the acute stage should be easily digestible and nutritious. Care should be taken to prevent deformity due to the unopposed contractions of healthy muscles, as when foot- or wrist-drop is present. This may be done by passive movements and mechanical appliance. Thus, foot-drop may be overcome by pads exerting pressure against the ball of the foot. If there is much hyperesthesia, measures to support the bed-clothing must be employed.

Rest in bed with the administration of strychnine and iron are indicated in diphtheritic neuritis. In that due to bad measures to increase the elimination of the lead, as small doses of iodide of potassium, purging with sulphate of magnesia and warm baths, must be employed. Massage and electricity are indicated in all forms of neuritis after acute inflammation has subsided.

CHAPTER VII

DISEASES OF THE CRANIAL NERVES

OLFACTORY NERVES AND TRACT

The functions of these nerves may be disturbed by disease anywhere in their course, from their distribution in the nasal mucous membrane to their centres in the cerebral cortex (uncinate gyrus). This disturbance may be manifested in subjective sensations of smell or parosmia; increased sensitiveness or hyperosmia; and loss of the sense of smell or anosmia. Loss of taste is frequently associated with the latter.

Parosmia.—Hallucinations of smell are found in the insane, in whom the sensation is usually unpleasant; in epileptics, in whom sometimes the *aura* may be represented by an unpleasant odor; and in cases of tumor situated in the region of the cerebral centres for smell. Rarely after head injuries the sense is perverted, odors of different character alike, or the odor may be changed.

Hyperosmia.—This usually occurs in nervous, hysterical individuals.

Anosmia.—This may be produced by: 1. Affections of the "terminals" of the nerves in the nasal mucous membrane, with catarrhal diseases of which it is commonly associated. This is the most frequent cause.

2. Lesions of the bulbs or tracts, which may be due to falls or blows; caries of the cranial bones; meningitis or tumor. The sense of smell may be lost in locomotor ataxia, probably due to atrophy of the nerves.

3. Lesions of the olfactory centres. In some cases the loss may be congenital, owing to the centres not being developed.

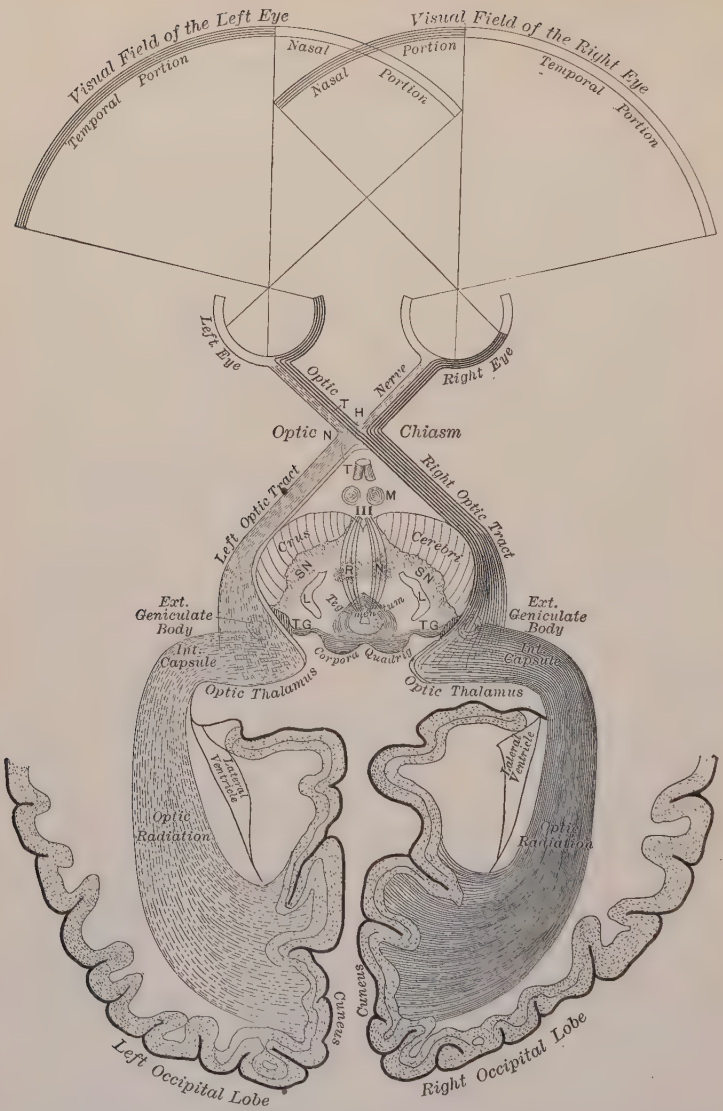
Tests for the sense of smell have been described on p. 59.

OPTIC NERVE AND TRACT

The Visual Pathway.—This commences in the rods and cones of each retina, the deepest layer of which gives origin to the optic nerves. These pass to the chiasm, where partial decussation occurs, and each optic tract as it leaves the chiasm contains fibers "originating" in the retina of each eye. Each nerve therefore contains three fasciculi: (1) A bundle of direct fibers which comes from the external or temporal third of each retina and passes into the optic tract of the same side; (2) a bundle of fibers which comes from the internal or nasal two-thirds of the retina, and at the chiasm passes into the optic tract of the opposite side; (3) a bundle of fibers which proceeds from the macular region and, decussating, passes partly to the optic tract of the same side and partly to that of the opposite side (Fig. 34). After leaving the chiasm each tract passes backward, winding around the cerebral crus, and most of the fibers end in the anterior quadrigeminal body, the lateral geniculate body, and the posterior part of the optic thalamus, or pulvinar. These constitute the primary optic centres, and are concerned in the movements of the eye and reflex movements of the iris and ciliary body. From these centres fibers arise which pass through the posterior part of the internal capsule, and, as the radiations of Gratiolet, pass to the region about the calcarine fissure in the occipital lobe (Figs. 82 and 83). The fibers from the macular region, according to Ferrier, pass to the angular gyrus of the opposite side, and lesions in this region have been found to cause amblyopia in the eye of the opposite side (crossed amblyopia). Other authorities (Henschen, von Monakow, and Harris) believe that the centre for macular representation is the occipital lobe (calcarine fissure, cuneus). It is probable that both regions are connected with macular vision, the angular gyri, especially of the left side, being particularly concerned with the power of reading.

In the calcarine region (primary cortical centre) therefore of each side are represented the temporal third of the retina

FIG. 34



The visual tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia: *H*, lesion at chiasm causing bilateral temporal hemianopsia; *N*, lesion at chiasm causing unilateral nasal hemianopsia; *T*, lesion at chiasm causing unilateral temporal hemianopsia; *SN*, substantial nigra of crus; *L*, lemniscus in crus; *RN*, red nucleus; *III*, third nerves. Macular bundles not shown.

of the same side and the nasal two-thirds of the opposite side. Each angular gyrus (higher or secondary cortical centre) is, according to Ferrier, in relation with the macula of the opposite side.

Amblyopia is a term used to signify dimness of vision. It is usually applied when no changes are visible in the eyeball to account for such loss. It is therefore due to either functional or organic disease of the retina, optic nerve or cortical visual centres. When complete loss of vision or blindness is so produced it is termed *amaurosis*.

Functional Disturbances of Vision.—Either functional amblyopia or amaurosis may be caused by hysteria (p. 463); the former is the more frequent. Amblyopia may also occur in neurasthenia (p. 447). Cases have been recorded due to reflex irritations, as decayed teeth.

Amblyopia may be caused by a number of poisons, as tobacco, alcohol, especially wood, lead, diabetes, uremia, various drugs, as quinine and salicylic acid, and infectious diseases. Amaurosis may be also so caused. Tobacco excessively used is the most common.

The loss of sight is gradual and equal in both eyes. The centre of the field of vision is particularly affected. Central scotomata (blind spots) are found for red and green. Changes in the fundus are frequently absent. The lesion in toxic cases is a retrobulbar neuritis. (See Optic Neuritis.)

Retinal hyperesthesia is sometimes seen in hysterical individuals.

Optic Neuritis.—Neuritis of the optic nerve occurs in two forms: first, in which there are *distinct lesions* at the intra-ocular end of the nerve (intra-ocular optic neuritis); and second, those cases which are unassociated with such lesions (orbital optic neuritis or retrobulbar neuritis).

The *first* is the form which principally concerns the neurologist. It is also known as *papillitis*. When there is great engorgement of the end of the nerve, so that it projects into the interior of the eye, the condition is spoken of as *choked disk* or *papilledema*.

Etiology.—The mechanism of the development of papilledema has caused much dispute, and according to the

cause may differ in different cases. That due to tumor of the brain, the most frequent cause, is believed by some[1] to be due to compression of the vessels as they pass through the lamina cribrosa or nerve; others[2] that it is "due to a venous engorgement that results from the rise of intravenous pressure that is necessary in order that circulation should be maintained in the intravaginal portion of the vein where this is subjected to an increased sheath pressure. The increased sheath pressure is also the origin of the second factor, the obstruction to the lymph drainage from the papilla." It seems evident that in tumor at least inflammation does not enter into the process. In those forms occurring in inflammatory conditions it may. A toxic factor may also be present in both tumor and inflammatory forms. The causes of papilledema and conditions which simulate it are brain tumor, abscess of the brain, meningitis, apoplexy, chronic hydrocephalus, thrombosis of the cavernous sinus, inflammation of the accessory sinuses (frontal, alveolar), nephritis, uremia, anemia, and disease of the orbital region.

Symptoms.—There is increased redness of the nerve head, the edges of which are hazy; the veins are dark, tortuous, and distended, and dip into the infiltrated tissue. The arteries are often smaller than normal. In severe forms flame-shaped hemorrhages may be seen. The surface is more or less elevated. In addition to these appearances, if choked disk exists, the nerve head is pushed forward into the interior of the eye, and the retinal vessels may be hidden in places. Vision may be unaffected until late in the disease. Occasionally it is lost rapidly.

Inflammation of the nerve or optic neuritis may be due to meningitis, syphilis, acute infectious diseases, lead, alcohol, rheumatism, and exposure to cold; marked elevation of the nerve head is not usually present from these causes.

Treatment.—The treatment of optic neuritis comes within the province of the ophthalmologist. When due to intracranial causes the ultimate result depends whether the cause

[1] Cushing and Bordley, Jour. Amer. Med. Assoc., 1909, lii, 353.
[2] Paton and Holmes, Brain, 1911, p. 389.

can be relieved or not. The operation of "decompression" (p. 363) frequently causes relief when the cause is increased intracranial pressure.

Optic Atrophy.—Etiology.—This may be *primary*, most of the cases of which are associated with disease of the spinal cord and brain, notably locomotor ataxia, paretic dementia, and disseminated sclerosis. Cold, infectious diseases, sexual excess, diabetes, arteriosclerosis, alcohol, and lead have also been assigned as causes of this form. There is also an hereditary form. (See Amaurotic Family Idiocy.)

There is also *secondary atrophy*, which results from a pressure more or less directly applied to the optic nerve chiasm or tracts.

Consecutive atrophy follows either neuritis or papilledema.

Symptoms.—These are loss of sight, indicated by diminished acuity of vision, diminution of the visual field, and altered perception of color.

In *primary atrophy* the disk has a gray tint. In *secondary atrophy* it is white in color.

Hemianopsia.—A lesion in the optic **chiasm, tract,** or **centres** produces hemianopsia (p. 58).

This is divided into *horizontal* when the dividing line is horizontal, and *vertical* when the dividing line is vertical.

Horizontal hemianopsia can be caused by a lesion so situated as to press upon the upper or lower part of the chiasm, downward upon one optic tract, or upon the upper or lower part of both optic nerves.

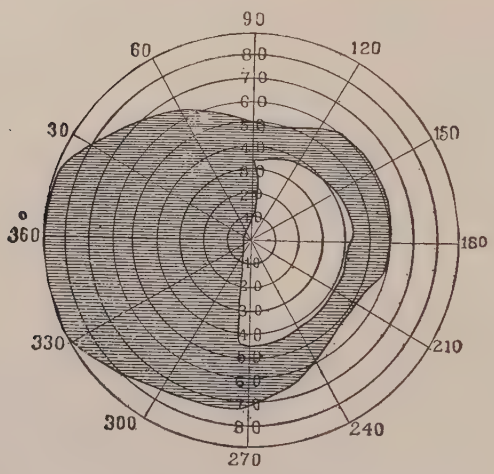
Vertical Hemianopsia.—This is subdivided into several varieties:

Bitemporal hemianopsia (Fig. 35), in which both temporal fields are wanting, can only be caused by a lesion involving the crossing fibers of both optic tracts in the middle of the chiasm (Fig. 34).

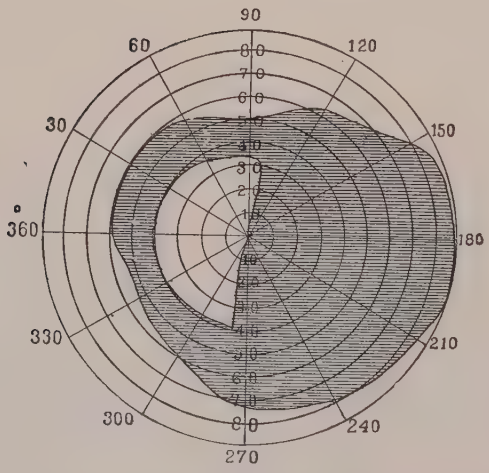
Binasal hemianopsia, in which both nasal fields are wanting, is rare. A lesion to produce this must be on both sides of the chiasm; or two lesions, one on the outer side of each optic nerve, are required (Fig. 34).

Homonymous hemianopsia is that in which the corresponding half of the visual field in each eye is wanting.

FIG. 35



Left eye.

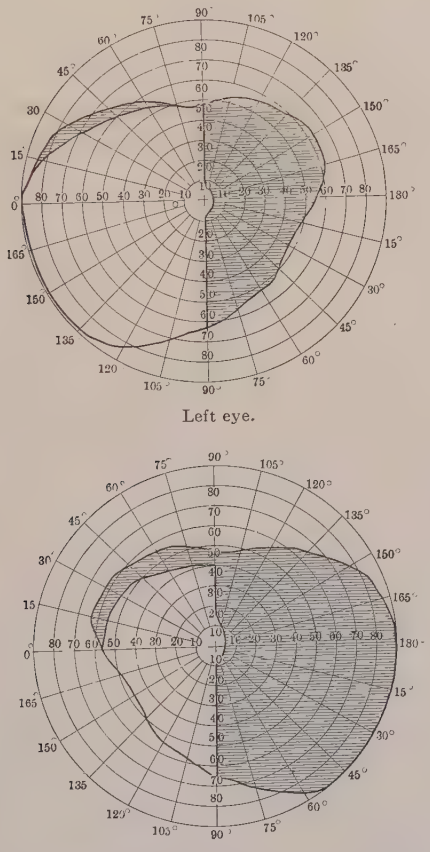


Right eye.

Bitemporal hemianopsia from a case of acromegaly originally under the care of H. C. Wood and later studied by F. A. Packard. Eyes examined in 1885 by de Schweinitz and above fields found.

If both right halves are wanting it is called a right homonymous lateral hemianopsia (Fig. 36); if both left halves, a left homonymous lateral hemianopsia. The loss

FIG. 36



Left eye.

Right eye.

Right homonymous hemianopsia, from a patient under the care of Wharton Sinkler.

of function is always in the opposite side of the retina from the blind field. Thus, if the left halves of the fields are wanting, the right halves of the retina are blind (Fig. 34).

This form of hemianopsia may be produced by a lesion situated in the visual tract posterior to the chiasm—*i. e.*, in the occipital lobe involving the cortical centres of vision; in the optic radiations, internal capsule—primary optic centres or optic tract (Fig. 34). The lesion is on the opposite side to the blind fields. It is either in the nerve or in the primary optic centres—consequently, interfering with the reflex arc of the pupil—if Wernicke's symptom is present (p. 65).

Homonymous hemianopsia is the most common form. It may be complete, the entire half field with the exception of the small area at the centre (macular vision), where the visual area extends a little to the far side of the dividing line (Fig. 36), being wanting or incomplete. It may be absolute—that is, all three functions of sight, viz., perception of light, of form, and of color are wanting; or it may be relative—that is, light sense is preserved and one or both of the other forms are wanting in the deficient area of the field.

Relative hemianopsia is always due to a cortical lesion; but cortical lesions may produce the absolute form. In cortical lesions there is usually contraction of the preserved half field, the contraction being more marked in that of the eye opposite the lesion. More rarely this may be due to a lesion of the tract. Homonymous hemianopsia may be due to functional causes, and occurs infrequently in hysteria and migraine. In such cases it is usually transient. An organic lesion near the visual tract may also cause a transient hemianopsia.

Visual Fields.—These may be contracted in disease of the optic nerve and also in functional diseases (p. 466). The color fields may give important information, normally after white, the largest field is for blue, red is next, then green. In some diseases this order may be reversed, red being greater than blue and so on (Fig. 126). The symptom is known as *dyschromatopsia*. It occurs in hysteria (p. 467), and also some organic diseases of the brain, notably tumor.

Acromatopsia is inability to recognize colors (color blindness).

MOTOR NERVES OF THE EYEBALL

The motor nerves of the eyeball are the third or oculomotor; the fourth or pathetic; and the sixth or abducens. These nerves may be diseased separately or collectively.

The Third Nerve.—Anatomy.—The nucleus of origin of the third nerve consists of a number of distinct nests of cells, each of which is the nucleus which furnishes the nerve fibers for a certain muscle. The arrangement of these cells and the muscles they supply are, from before backward: sphincter iridis, ciliary muscle, convergence centre, rectus superior, rectus internus, levator palpebræ superioris, obliquus inferior, and rectus inferior. These are situated along the floor of the aqueduct of Sylvius beneath the corpora quadrigemina (Fig. 38). From there the nerve passes through the crus, at the side of which it emerges. After passing along the cavernous sinus it enters the orbit through the sphenoidal fissure and supplies, by its superior branch, the levator palpebræ superioris and superior rectus; by its inferior branch, the inferior oblique and internal and inferior recti muscles. Other branches supply the ciliary muscle and constrictor of the iris.

Paralysis of the Muscles Supplied by the Third Nerve.—Etiology.—A common cause is injury to the nerve either by pressure or neuritis. A meningitis, usually syphilitic, is the most common cause of this, but it may be due to other forms, especially the tubercular. Other causes are those causing multiple neuritis (p. 124), especially diphtheria; arteriosclerosis (p. 120); traumatism, causing either fracture of the base of the skull or hemorrhage there; transitory forms may be due to exposure (rheumatic); tumor of the brain; tumor within the orbit or anything causing marked exophthalmos; and after spinal anesthesia.[1]

Degeneration of the nucleus due to toxemia (p. 150) may be a cause. This also occurs in tabes dorsalis, paresis, and disseminated sclerosis.

[1] The sixth is more commonly involved. Vide Reber, Jour. Amer. Med. Assoc., July 30, 1910, p. 380.

Symptoms.—In total *third nerve paralysis* the upper lid hangs down, almost or completely covering the eyeball (ptosis). On opening the eyelids with the fingers the eye is noticed to be turned outward and slightly downward (action of external rectus and superior oblique), and the pupil is dilated and responds neither to light nor accommodation. The eye may project slightly, owing to the weakness of most of the muscles which retain it in place. Diplopia (p. 56) is complained of (Fig. 37). There is no loss of sensation in the conjunctiva. Cases are recorded in which the paralysis was limited to the extra-ocular muscles, the iris and ciliary

FIG. 37

Paralysis of the left third cranial nerve: 1, ptosis; 2, ineffectual attempt of frontalis to overcome ptosis; 3 shows partial ptosis and outward deviation of affected eyeball. (Church and Peterson.)

muscle escaping. Such cases are probably due either to a lesion situated at the beginning of the peripheral course of the nerve, just before all the fibers have converged into one trunk, the ciliary and pupillary fibers being there nearer the middle line; to a lesion attacking the branches of the trunk within the orbit, after they have divided to go to the various muscles, or to a nuclear lesion (p. 150).

Owing to similar situations of the lesion there may be paralysis limited to a single extra-ocular muscle. That of the *internal rectus* is the most common. When the muscle of one side is affected the patient holds his head downward and toward the paralyzed side.

Isolated paralysis of the *levator palpebræ superioris*, causing ptosis, may be due, in addition to a lesion involving the branches as above mentioned, to a disease of the nucleus, or to a lesion situated in the tract of the third nerve between the nucleus and its cortical centre. It, however, is often the first symptom of a later more complete third-nerve palsy, in some instances preceding for weeks the involvement of the other muscles. Isolated paralysis of the *inferior oblique* is exceedingly uncommon.

The paralysis may be limited to the *intra-ocular muscles*. If complete, there would be dilatation and immobility of the pupil (*iridoplegia*) to both light and accommodation and convergence; when there is loss of response to light only it is called reflex iridoplegia; when there is loss of the power of accommodation and convergence only, it is termed *cycloplegia*. Either one of these functions may be affected without the other. The lesion is usually a nuclear degeneration. Reflex iridoplegia (Argyll-Robertson pupil) is most frequently seen in locomotor ataxia and paretic dementia. Total iridoplegia is most often seen in cerebral syphilis. Either may be the permanent symptom of a former more extensive third-nerve palsy. A bilateral paralysis of the third nerve is most frequently syphilitic.

Recurrent Form.—This affection, also known as *ophthalmoplegic migraine* (p. 107) and periodic ophthalmoplegia, is rare. It most frequently attacks females. It usually begins early in life. The symptoms are unilateral headache, lassitude, nausea, slight fever, and complete paralysis of the third nerve on the same side as the pain. Other cranial nerves (fifth, sixth, and seventh) may be involved. The attacks come periodically and last from several days to long periods of time. In some cases one or more of the muscles do not recover, but remain permanently paralyzed.

The *pathology* is uncertain.

The Fourth Nerve.—Anatomy.—The fourth nerve originates from a nucleus at the level of the depression midway between the anterior and posterior corpora quadrigemina. It lies near the middle line in front of the gray matter which surrounds the aqueduct of Sylvius, in a hollow on the dorsal

side of the posterior longitudinal fasciculus; and is separated by a very slight interval from the lower end of the third nucleus (Bruce) (Figs. 38 and 39). Most of the fibers after leaving the nucleus decussate in the valve of Vieussens. The external origin of the nerve is from the upper surface of this valve, whence it passes around the crus to the anterior edge of the pons. From there it passes along the cavernous sinus, enters the orbit through the sphenoidal fissure, and supplies the superior oblique muscle.

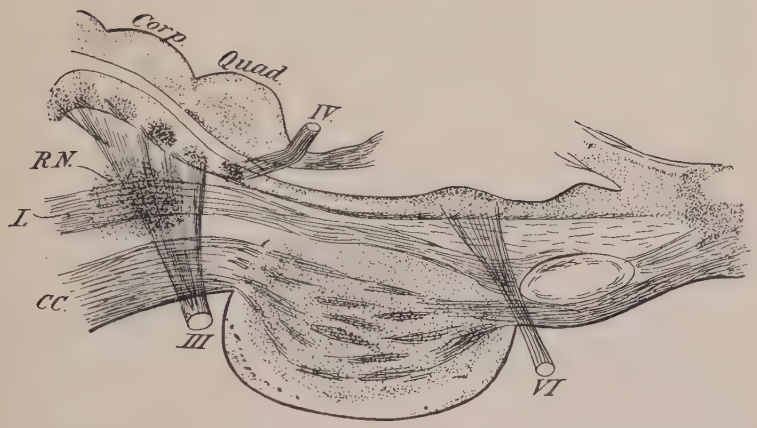
Paralysis of the Muscle Supplied by the Fourth Nerve.—Symptoms.—Isolated paralysis of the superior oblique muscle is most rare. When present the eye is rotated upward and inward. On looking downward there is slight turning inward of the eye with homonymous diplopia (p. 56) in the lower half of the field and convergent strabismus. Marked vertigo is also a frequent symptom.

Involvement of the nerve trunk or its nucleus due to causes similar to those producing paralysis of the third nerve are the *etiological factors*.

The Sixth Nerve.—Anatomy.—The sixth nerve arises from a nucleus in the floor of the fourth ventricle (Fig. 38). It is bordered upon its inferior, upper, and inner sides by the root of the facial nerve. Its superficial origin is between the pons and medulla, external to the pyramids. It enters the wall of the cavernous sinus, passes through the sphenoidal fissure to the orbit, and supplies the external rectus muscle. Its nucleus is connected with those of the third and fourth nerves by the posterior longitudinal fasciculus. Fibers from the sixth nucleus also pass through the fasciculus to the nuclei of the third nerve (that of the internal rectus muscle) on the opposite side (Fig. 39). Through this the sixth nucleus governs conjugate deviation of the eyes, *i. e.*, when the internal rectus of the right eye and the external of the left cause both eyes to look to the left side, or *vice versa*.

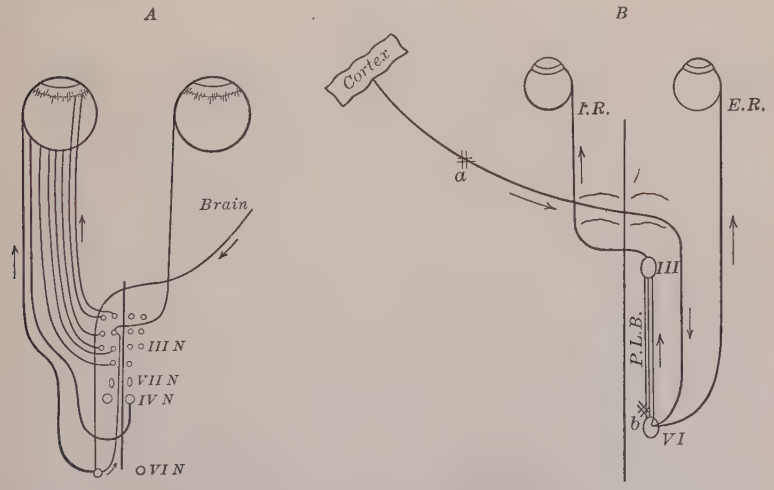
Loss of lateral associated movements is due to a lesion involving either the sixth nucleus or the posterior longitudinal fasciculus; it is therefore found in intrapontine lesions. Loss of associated movements of the eyes upward indicates a lesion in the region of the aqueduct of Sylvius.

Fig. 38



Sagittal section through the cerebral axis, to show the nuclei of the ocular nerves in the floor of the aqueduct of Sylvius and the fourth ventricle, and the course of the nerves to their exit. The various groups of cells from which the third nerve arises are seen: *RN*, red nucleus of tegmentum; *L*, lemniscus (sensory tract); *CC*, motor tract in the crus cerebri seen to traverse the pons and enter the anterior pyramid of the medulla. (Starr.)

Fig. 39



A, diagram showing the arrangement of the nuclei of the motor nerves of the eye, and the decussation of the fourth and internal rectus branch of the third nerve.

B, diagram showing the probable relations of the nuclei of the sixth and of the internal rectus branch of the third to the brain. *P.L.B.*, posterior longitudinal bundle.

The cortical centres of these muscles are not definitely known; they may possibly be in the second frontal convolution (Fig. 84). The fibers from these pass through the internal capsule, those of the third at the "knee," before reaching the nuclei. Most of those which pass to the third and sixth nuclei decussate, and terminate in the nuclei of the opposite side. As has already been said, the fibers of the fourth nerve do not decussate until after they have left the nucleus to pass to the periphery.

Paralysis of the Muscles Supplied by the Sixth Nerve.—Symptoms and Etiology.—Paralysis of the external rectus muscles causes either a convergent strabismus or in mild cases inability to make the involved eye look to the paralyzed side, with diplopia (p. 56). Vertigo may be a symptom. It is due to disease either of the nerve trunk or its nucleus, the causes of which are similar to those producing interference with the functions of the third and fourth nerves (p. 143). It is at times an early symptom of tabes dorsalis. In old age transient weakness due to arteriosclerosis has been observed. Brain tumor not involving either the nucleus, intrapontine or peripheral fibers may cause paralysis of this nerve (p. 359). There is also a double palsy which is congenital.

Combined Paralysis of the Ocular Muscles.—Etiology.—Paralysis of the third, fourth, and sixth nerves may be caused by a lesion of the cortex, nuclei, or nerve trunks. It is most commonly due to a gross lesion at the base of the brain, as meningitis, most frequently syphilitic or tuberculous; tumor, or fracture involving the sphenoidal fissure or cavernous sinus. It may be due to *thrombosis of the cavernous sinus,* usually due to some septic process in the brain or its vicinity (p. 338), when it will be associated with exophthalmos, dilatation of the frontal veins, cyanosis and edema of the orbital and frontal regions. While paralysis of any ocular muscle due to any cause is an *ophthalmoplegia,* the term is more commonly applied when due to a *nuclear disease.* This may be acute or chronic.

Acute Ophthalmoplegia.—Etiology.—It may be caused by a hemorrhage from the small arteries supplying the nuclei.

This is usually traumatic. Thrombosis or embolism in these arteries may also cause it. Acute hemorrhagic polio-encephalitis (p. 336) involving the nuclei (polio-encephalitis superior of Wernicke) may produce it. The causes of the latter are the infectious febrile diseases, especially influenza; tuberculosis; possibly syphilis; alcohol; ptomains from decayed fish and meat; and lead. Toxic forms, especially due to ptomains, occur in which there is no anatomical lesion. A myositis of the ocular muscles due to rheumatism may simulate it.

Symptoms.—When due to hemorrhage there would be a sudden paralysis of the ocular muscles, preceded probably by vomiting and convulsions. When an acute hemorrhagic polio-encephalitis is the lesion there may be prodromes, such as general malaise, headache, vertigo, and vomiting. The temperature may be either slightly elevated, subnormal, or remain normal. Soon there is rapidly developing paralysis of the ocular muscles, excepting the levator palpebræ superioris and sphincter of the iris, which often escape. Headache and vertigo continue, and soon stupor and in severe cases coma occur. Acute ophthalmoplegia is usually bilateral, and may be associated with poliomyelitis, and with paralysis of muscles of the face, tongue, and palate, due to involvement of their nuclei (bulbar palsy).

Diagnosis and Prognosis.—The presence of convulsions, muscular twitchings, headache, stupor, or coma would distinguish the affection from neuritis, and it must be remembered that the affection may be due to neuritis. The distinction may often be difficult to make, but absence of the symptoms above mentioned and the existence of neuritis of other nerves would be in favor of such as the cause of the trouble. The neuritic form has a more favorable prognosis. It may also have to be distinguished from myasthenia gravis (p. 267). The disease is often fatal, the most favorable cases being those which follow the infectious diseases. In cases which recover paralysis may remain (chronic ophthalmoplegia).

Treatment.—That of acute hemorrhagic encephalitis. (See p. 338.) Syphilis if suspected as a cause should be vigorously treated.

Chronic Ophthalmoplegia.—Etiology.—Chronic ophthalmoplegia, or chronic nuclear ocular paralysis, may occur as (1) one of the terminations of the acute, the lesions of which have produced degenerative changes; (2) a chronic degeneration of the nuclei (chronic polio-encephalitis superior) which may be due to syphilis, diphtheria, diabetes, or be in association with or preceding the development of tabes, paresis, disseminated sclerosis, progressive muscular atrophy, chronic bulbar palsy (chronic polio-encephalitis inferior); (3) a congenital and sometimes hereditary affection. The lesion is a chronic degenerative or inflammatory process found in the floor of the third ventricle and aqueduct of Sylvius, and which causes atrophy of the ganglion cells of the nuclei there situated. The process is similar to that occurring in either progressive spinal muscular atrophy or chronic poliomyelitis.

Symptoms.—The affection is characterized by a gradual and progressive paralysis of many or all of the ocular muscles. Often one of the first symptoms complained of is diplopia, which may be transient. The other muscles then become involved, often in an irregular manner, without reference to their functions. Ptosis may be either absent or is incomplete. The paralysis may progress until a certain development is reached and then remain stationary; or it may continue until all of the muscles are involved. Both eyes are usually affected, but it may be unilateral. Diplopia is apt to disappear in the later stages. In some cases the intra-ocular muscles escape (ophthalmoplegia externa); in others they are the only ones to become affected (ophthalmoplegia interna).

Diagnosis.—The distinction must be made between paralysis due to peripheral neuritis, disease at the base of the brain, and intracerebral disease. This at times may be difficult. From neuritis, nuclear disease differs in the absence or slight development of ptosis, the bilateral distribution of the symptoms, and the association of the palsy, with symptoms of degenerative cerebral or spinal disease. The exemption of the intra-ocular muscles, when present, is in favor of nuclear disease; but it may occur in neuritis. In disease

at the base, headache, nausea, vertigo, and optic neuritis would probably be present. From the close proximity of their trunks at the base of the brain the fifth nerve would probably suffer with the sixth, and other cranial nerves would be apt to be irregularly involved (Fig. 97).

A lesion of the *crus cerebri* would probably cause paralysis of the third nerve, which would be associated with hemiplegia of the opposite side. Lesions in the region of the corpora quadrigemina may also cause paralysis of the third nerve, owing to the closeness of its nuclei to these structures (Fig. 38) (p. 146). Such paralysis would be accompanied by incoördination of movement, and possibly forced movements. If the lesion is a tumor, general symptoms common to cerebral growths occur.

Ptosis may be produced by disease of the sympathetic, causing paralysis of the smooth muscular fibers in the fascia of the orbit (fibers of Müller). In these cases, which are uncommon, the ptosis is incomplete, and there would be contraction of the pupil upon the diseased side.

Prognosis.—If the primary cause, such as syphilis or rheumatism, can be influenced by treatment the prognosis is good; but it must be remembered that even when due to these, permanent damage may have been done to the nuclei or nerve fibers, when permanent paralysis would result. When a symptom or forerunner of degenerative disease of other portions of the nervous system (tabes, etc.), the possibility of which should always be remembered, the prognosis is not good, although sometimes the paralysis may disappear.

Treatment.—Treatment consists of the use of potassium iodide and mercury in large doses when a history of syphilis is obtained, and even when no history of such is given it is well to give these drugs a thorough trial, as syphilis is a most frequent cause of such palsies either single or combined. If, in syphilitic cases, the symptoms are urgent salvarsan may be of service. Salicylates should be administered if there is a history of rheumatism. Tonics, such as strychnine, arsenic, and small doses of bichloride of mercury, may be tried. A weak (1 milliampère) constant galvanic

current, the positive pole at the nape of the neck and the negative over the affected eye, is also useful. When it exists as a complication of some other disease treatment for that disease should be employed. Diplopia may be corrected by employment of prisms, or by placing a ground-glass over one eye.

Spasms of the Ocular Muscles.—Etiology and Symptoms.—These may be tonic or clonic; in origin, functional or organic. The causes of functional spasm are hysteria, neurasthenia, and refractive errors. Tonic spasm of the orbicularis palpebrarum occurring in hysteria may simulate ptosis. A convergent strabismus may also be due to hysterical spasm of the internal recti. Other symptoms of hysteria would, of course, accompany these conditions (p. 460). Refractive errors are a common cause of ciliary spasm.

Spasm of organic origin is due to irritation in the course of the ocular nerves between the cortex and muscle. Conjugate deviation is the common manifestation of this spasm. This may be caused by irritation of any part of the cerebrum, but most markedly so when the irritation is near the motor centres of the limbs. The cortical centre for lateral movements of the eyes has been placed in the posterior part of the second frontal convolution (Fig. 82). Lesions in the region of the internal capsule also cause it. In spasmodic conjugate deviation in these situations the eyes look away from the side of the lesion; in the paralytic form, caused by a destructive lesion, the opposite is the case. Lesions in the pons also may produce this symptom; but here irritative lesions (spasm) being on the same side as the sixth nerve nucleus cause the eyes to deviate toward the side of the lesion; while destructive lesions (paralytic) cause the opposite effect (Fig. 39, *B*). Apparent spasm may occur in some of the manifestations of tic (p. 430).

Nystagmus is characterized by rapid, involuntary movements of the eyeball, usually from side to side, but sometimes in a vertical or rotary direction. It may only be seen when the patient is made to move the eyes in different directions, and then may only occur during certain move-

ments. In most cases it is bilateral. It may be congenital. It occurs in some eye diseases and in certain occupations. It is a common symptom of cerebellar tumor, disseminated sclerosis and Friedreich's ataxia. It may accompany various forms of paralytic and spasmodic disorder of ocular movements, due either to disease of the nuclei or of the commissural and association tracts which connect these nuclei and various parts of the brain together. It has also occurred in conjunction with disease of the internal ear p. 169. It is frequently present in blindness.

THE FIFTH NERVE

Anatomy.—The fifth or trigeminal nerve consists of two portions—a motor and a sensory. The central neurone for the motor portion arises in the lower extremity of the precentral convolution, near those for the lips and tongue. It passes from there downward, through the knee of the capsule, and after decussating joins the motor nucleus in the medulla. Fibers from this nucleus form the motor root, which eventually becomes part of the inferior maxillary division.

The sensory portion arises in the cells of the Gasserian ganglion,[1] the cells of which give off a long dendrite, from which the axone originates. The former goes to the periphery, forming the three divisions: ophthalmic, superior maxillary, and inferior maxillary of the sensory portion of the nerve.

The axones enter the pons, where they divide: one division (the ascending root) passes upward almost to the third-nerve nucleus. They terminate in free ramifications in the substantia gelatinosa. The other and largest division (descending root) passes downward into the cord about as far as the second cervical segment. These fibers end about cells in the substantia gelatinosa. From these, fibers arise which enter the fillet, decussate, and ascend to the brain

[1] The Gasserian ganglion is analogous to the ganglion upon the posterior roots of the spinal nerves.

cortex (central convolutions?). Collaterals from the descending root pass to the nuclei of the motor cranial nerves. Its many connections explain the liability of the nerve to reflex irritations.

FIG. 40

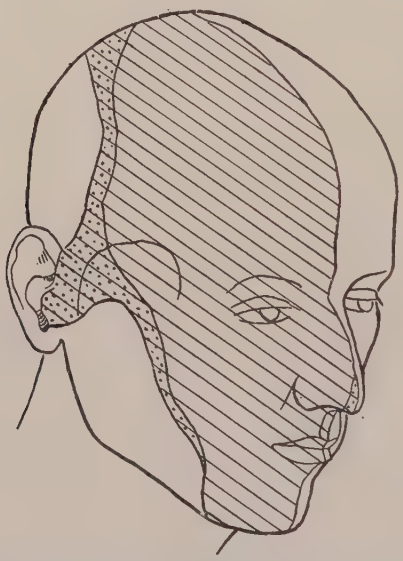


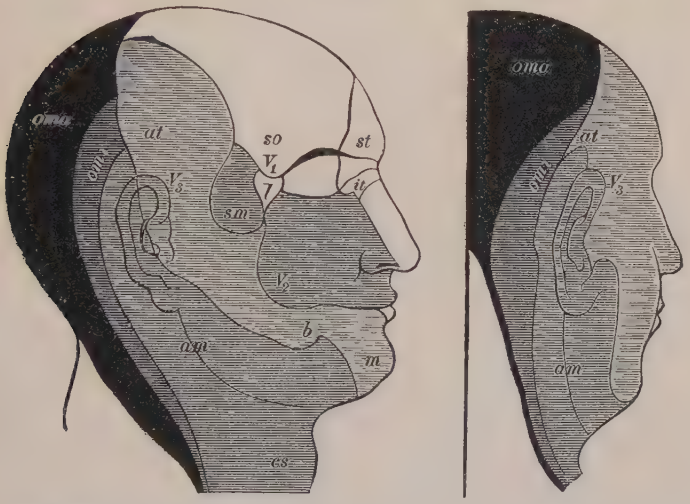
Diagram showing the normal (avèrage) field of postoperative cutaneous anesthesia. The shaded area, including the tragus and anterior wall of meatus, remains anesthetic to tactual (hair esthesiometer) stimuli. The dotted strip gives the impression of touch or pressure to pain stimuli (needle), with few if any actual pain points (Cushing, in Bull. Johns Hopkins Hosp., July-August, 1904).

The *sensory divisions* supply the skin of the face and head as far back as the occiput, most of the dura (p. 101), the conjunctiva and mucous membrane of the mouth, tongue,[1] upper part of pharynx, nose, and teeth (Figs. 40 and 41). Secretory fibers possibly go to the salivary and lacrymal glands. The *motor portion* supplies the tensor tympani

[1] A commonly accepted view is that sensations of taste from the anterior two-thirds of the tongue are conducted by the fifth nerve. Late observations after destruction of the ganglion seem to disprove this (p. 170). See Davies, Brain, 1907, p. 219.

and muscles of mastication, *i. e.*, masseters, pterygoids, temporals, mylohyoid, and anterior bulb of the digastric.

FIG. 41



Distribution of the sensory cutaneous nerves on the head: V_1, V_2, V_3, the three branches of the trigeminus; *at*, auriculotemporal; *so*, supraorbital; *st*, supratrochlear; *it*, infratrochlear; *l*, lacrymal; *m*, mental; *b*, buccinator; *am*, auricularis magnus, *sm*, subcutaneous malæ; *oma*, occipitalis major; *omi*, occipitalis minor; *cs*, superficial cervical. (After Hirt.)

Paralysis of the Fifth Nerve.—Etiology.—Either the motor, or the sensory root, or one of the divisions of the latter, owing to the difference of their course, may be affected or the entire nerve may be involved. A lesion situated at the base of the brain involving the superficial origin would cause a general paralysis, while a disease of the inferior maxillary division would cause motor and sensory symptoms, the latter being limited to the peripheral distribution of that branch.

The *chief causes* of paralysis of the nerve are as follows:

(*a*) The ophthalmic division lies in the cavernous sinus, where it may be damaged by tumors in the pituitary region. It may suffer along with the third, fourth, and sixth nerves

in thrombosis of the cavernous sinus (p. 148), and within the orbit it may be injured by newgrowths and inflammatory processes. The superior and inferior maxillary divisions lie in the sphenomaxillary fossa, where they are liable to suffer from wounds and tumors which may invade this region, and also from tumors on the floor of the middle cranial fossa.

(*b*) At the base of the brain the nerve may suffer from tumors, meningitis (especially syphilitic), and caries of the temporal bone.

(*c*) Disease in the pons, as tumor, hemorrhage, or softening, or a patch of sclerosis (in disseminated sclerosis), may damage the sensory nucleus (descending root). As a rule, owing to its great extent, damage to this nucleus causes only partial paralysis. Tumor in the cerebellopontine angle may involve this nerve.

(*d*) Traumatism to the mouth and nose may injure some branches of the nerve.

(*e*) Neuritis may occur in syphilitic, gouty, or rheumatic individuals, or from exposure, but is rare.

Symptoms.—Paralysis of the sensory division causes *loss of sensation* in the parts, both skin and mucous membranes, supplied by the nerve. The loss may affect the entire region when the disease is at the root, or if all three branches are diseased; but if only one branch is diseased, the loss is limited to the part supplied by it (Fig. 41). Pain sense is usually lost before the tactile.

Neuralgic pain in the course of the nerve often precedes the development of the anesthesia. Loss of taste in the anterior two-thirds of the tongue may be present early in the course of the disease or during the first few days after the ganglion is removed, but usually soon returns (p. 170). Various trophic and vasomotor symptoms may also result. Increase of the salivary and lacrymal secretions may occur if there is irritation; but when destruction has occurred, diminution.[1] Paleness of the face upon the affected side may

[1] Davies (loc. cit.) states that after neurectomy vasomotor and secretory symptoms do not occur.

be observed. The cornea often becomes dry and opaque, and ulceration and eventually destruction of the eyeball may result. Ocular changes are especially frequent in disease of the Gasserian ganglion or of the nerve anterior to it. Herpes, most often in the superior maxillary distribution, may be a symptom. In hemiatrophy of the face degeneration of the ascending root has been found.

The symptoms of disease of the *motor portion* are weakness or paralysis of the muscles of mastication upon the side of the lesion (p. 44).

Atrophy of the muscles may cause sinking in the temporal and zygomatic fossæ.

Diagnosis.—Complete paralysis, both motor and sensory, of the fifth nerve, does not resemble any other condition. In hemianesthesia there is anesthesia of the face in the distribution of the fifth nerve; but there is also loss of sensibility in the back of the head and of the trunk and limbs of one side, and often there is hemianopsia. When there is pain the condition might be confounded with neuralgia, but the existence of anesthesia would render plain the nature of the affection. The diagnosis of the *locality* of the lesion is important, and depends upon the distribution of the anesthesia and the accompanying symptoms.

Paralysis of the entire nerve, motor and sensory, associated with neuralgic pain is most probably due to disease at the base of the brain (cerebellopontine angle) or of the Gasserian ganglion. If there is also paralysis of the sixth nerve, the lesion would involve the pons. When the ophthalmic is alone affected, especially if there coexists paralysis of the motor nerves of the eyeball, the lesion is probably at the sphenoidal fissure or in the orbit; paralysis limited to the distribution of the superior maxillary points to a lesion in the superior maxillary bone or sphenomaxillary fissure.

Crossed paralysis of the fifth nerve is due to a lesion in the lower half of the pons or upper part of the medulla (Fig. 100). Conjugate deviation of the eyes from the side of the lesion associated with fifth-nerve paralysis is always due to a lesion in the pons.

Treatment.—Our success in treating paralysis of the fifth nerve, and consequently the *prognosis*, depends upon the cause. If it cannot be removed, nothing can be done. Antisyphilitic treatment should be tried. If a tumor, surgery may be of assistance. The application of heat is useful in cases due to exposure to cold and consequent neuritis. Counterirritation over the mastoid is also useful. For the relief of pain, in addition to heat, phenacetin, antipyrin, and similar remedies may be tried. The anesthesia, if the nerve has not been entirely destroyed, is often benefited by the frequent application of a rapidly interrupted faradic current applied through the wire brush. The eye should be kept thoroughly and frequently cleansed with boric-acid solution. In the intervals it is well to keep it covered with a watch-crystal held in place by adhesive strips.

Neuralgia of the Fifth Nerve.—Etiology.—The causes of neuralgia are stated on page 109. There is a special form of neuralgia of the fifth nerve known as *tic douloureux* or *prosopalgia*, which comes on at the degenerative period of life, after forty.

Symptoms.—The pain of neuralgia of the fifth nerve resembles in character that of neuralgia elsewhere (p. 109). It is also usually unilateral, deep seated, and corresponds to the distribution of the division or branch affected. It more commonly affects one or two divisions of the nerve than all three. During the paroxysms the pain is more intense at certain points, which are tender; and if the attack is of long duration, the tenderness remains between the paroxysms.

Neuralgia of the *ophthalmic division* is most usually confined to the supra-orbital branch, sometimes termed "brow ague," owing to the fact that it, more frequently than other forms, is due to malaria. The pain radiates over the front of the head from the supra-orbital notch, and may also be felt in the eyelid, in the eyeball, and high up on the side of the nose. A tender point usually exists at or near the supra-orbital notch. More rarely the tender points may be found on the upper eyelid and at the junction of the nasal bone with the cartilage. Ocular neuralgia, pain referred

to the eyeball, is also a common form. The pain may be spontaneous, but is often excited by the use of the eyes, and may be accompanied by dimness of vision and lacrymation. The pain feels as if it were deep in the orbit and is of a tearing character; it is sometimes bilateral. Those who have had rheumatic iritis are especially liable.

In neuralgia of the *superior maxillary division* the pain is felt in the face between the orbit and the mouth, in the side of the nose, and upper teeth. Tender points are found at the infra-orbital foramen, along the gum of the upper jaw, at the side of the nose, and over the prominent part of the malar bone.

When the *inferior maxillary division* is affected the pain may be felt just in front of the ear; in the posterior part of the temple; at the emergence of the nerve from the inferior dental foramen; just above the parietal eminence, and at the side of the tongue. The pain may be confined to one of these regions or may be diffused, involving all of these points. Movements of the jaw or tongue are liable to aggravate the paroxysms in this form.

The pain may at times extend to other branches than the one affected or to other nerves. In very severe neuralgia there may occur reflex twitchings of the facial muscles during the paroxysms. There may be vasomotor disturbances, as congestion, flushings, and sweating. Trophic symptoms, especially herpes, may also occur.

Tic douloureux occurring after forty is chronic in its course, and is especially apt to affect the superior maxillary division, but may affect two or all; is characterized by most agonizing pain, which occurs suddenly and frequently, but is of short duration, and is usually accompanied by facial spasm. The morbid anatomy of neuralgia is discussed on (p. 109).

Diagnosis.—The points by which we would diagnose neuralgia from neuritis and headache, with which it would be most liable to be confounded, have been mentioned on page 104. Having determined that the condition does exist, it is essential to determine the cause if possible. Pains in the course of this nerve may also be caused by cerebello-

pontile growths (p. 356), and disease of the Gasserian ganglion, in this event anesthesia will coexist (p. 157).

Prognosis.—When there is a cause that can be removed, such as anemia or debility, the prognosis is good. In cases due to any cause of long duration the possibility of degenerative changes occurring in the nerve must be considered. The longer the duration the worse the prognosis. The forms occurring after middle life are exceedingly intractable.

Neuralgia, no matter how severe, does not apparently shorten life.

Treatment.—This consists of measures for the relief of pain and for the removal of the cause. The salicylates are most useful when the trouble is rheumatic, and may be tried in any case. They must be given in full doses (gr. x–xv, four times daily). Antipyrin, phenacetin, and similar remedies are most useful, but must be used with caution, remembering their depressing action upon the heart. In neurotic individuals small doses of the bromides may give relief. Nitroglycerin (gr. $\frac{1}{100}$ every hour) is useful, especially in neuralgias of the aged. Alcohol, valerian, or compound spirit of ether may abort an attack. Atropine hypodermically is effective in some cases. One of the most valuable remedies is *aconitia*, which may be given in doses commencing with gr. $\frac{1}{200}$ and gradually increasing by gr. $\frac{1}{200}$ each time, when necessary, until the physiological action of the drug is produced. This drug in a number of cases is curative, and its use if watched may be continued a long time. In supra-orbital neuralgia, or in fact in any periodical neuralgia, large doses of quinine are often beneficial. Ergot in full doses has also been recommended for this form. If anemia exists iron and arsenic should be given.

The local application of dry heat, of liniments composed of equal parts of chloral and camphor, or of menthol (gr. xx–℥j), may give relief. A valuable means of affording relief is the use of electricity. It is best applied by placing the cathode at some indifferent point and placing the anode over the painful points in turn. The current should be constant, of moderate strength, which should be decreased gradually, and applied daily or oftener if possible. The

placing of a 20 per cent. solution of cocaine upon the electrode increases its effect (cataphoresis). *X*-rays have also been used with success.

To remove the cause all sources of reflex irritation must be sought for: carious teeth removed or put in good condition, catarrh of the nose or throat treated, and the digestive organs carefully looked after. The excessive use of tea, coffee, alcohol, tobacco, or sexual indulgence must be prohibited, and the patient put under as good hygienic conditions as possible. A removal to a warm, equable climate may bring relief. For *chronic cases* Dana has recently advocated a plan of treatment which sometimes proves successful: The patient is put to bed, given liquid diet, and once each day given a hypodermic injection of strychnine, beginning with gr. $\frac{1}{20}$ and gradually increasing until a dose of $\frac{1}{5}$ to $\frac{1}{4}$ gr., if possible, is reached. It takes about three weeks to do this. The dose is then gradually decreased until the original dose is reached. This entire process takes about six weeks, and should be repeated if the pain does not entirely disappear or a relapse occurs. During this time iron and iodide of potassium, or other appropriate drugs, may also be given. This seems to be of special service in patients who are debilitated. When the second or third branches are affected the injection of alcohol into the nerve at either the foramen rotundum or ovale, as the case may be, is the most satisfactory method of treatment for those which will not yield to ordinary measures. Relief lasting several years may be so obtained.[1]

When all else fails her, removal of the ganglion, or cutting the sensory root by the method of Frazier and Spiller, must be considered. The technique of these different operations is described in works on surgery. In severe cases, if relief is not secured, it is not well to wait too long before advocating surgical measures, for fear that degenerative changes may advance beyond the ganglion into the nuclei, when, of course, nothing can be done.

[1] The technique, indications, and contra-indications for this comparatively simple operation are well given by Patrick in Jour. Amer. Med. Assoc., January 20, 1912, p. 155.

THE SEVENTH NERVE

Anatomy.—The central neurones of the facial nerve arise from the cells of the lower extremity of the precentral convolution (Fig. 84). Fibers from these form a tract which passes through the corona radiata, the anterior portion of the posterior limb of the capsule, to the tegmentum, where they decussate and enter the nucleus of the opposite side. This is situated at the lower part of the pons, in a line with the motor nuclei of the fifth, ninth, and tenth nerves (nucleus ambiguus). To its outer side is the descending root of the fifth nerve. From the nucleus the fibers take a circuitous course, bending to form a V, in the bottom of which lies the nucleus of the sixth nerve. They emerge to form the facial nerve from the lower border of the pons, between the reflex extremity of the olive and the restiform body. The eighth or auditory nerve is separated but very slightly from it. The nerve may be subdivided into three portions: (1) An intracranial portion, which extends from the superficial origin to where it enters the internal auditory meatus; (2) an intra-osseous portion, which begins at the internal auditory meatus and runs through the Fallopian canal to the stylomastoid foramen; and (3) an extracranial portion, or that part of the nerve external to the stylomastoid foramen. Within the Fallopian canal, at what is known as the "knee" of the facial, is the geniculate ganglion. From this arise two nerves: the intermediary nerve of Wrisberg and the chorda tympani.

The *chorda tympani*, through which sensations of taste are transmitted, runs through the canal in company with the seventh nerve, and finally joins the lingual branch of the fifth to be distributed to the tongue. It is regarded by some as a continuation of the nerve of Wrisberg (p. 170).

Why in supranuclear palsy of the face the muscles of the upper part of the face escape is a disputed point. Possibly it may be due to the fact that they are muscles which usually act together and hence may be innervated from either side of the brain. That the oculomotor nucleus

is the origin of the upper facial nerve is disproved by late observations.[1] All of the fibers of the facial nerve probably arise from what is known as the facial nucleus.

The seventh nerve supplies all the muscles of the face, eyelids, and mouth, excepting those supplied by the third and fifth nerves; also the occipitofrontalis, platysma, the muscles of the eyebrows, the extrinsic muscles of the ear and of the nose, posterior belly of digastric, and the stylohyoid.

Paralysis of the Seventh Nerve.—Etiology.—Paralysis of the muscles supplied by the facial nerve may be due to lesions of either the central or peripheral neurone. If of the former, it may be either cortical or subcortical, in or near the internal capsule, or in the pons. If of the latter, it may be either nuclear or of the nerve trunk. The nerve itself may be involved in one of three situations, viz., at the base or intracranial portion, within the Fallopian canal, or intra-osseous portion, or external to the stylomastoid foramen, or extracranial portion.

Central or supranuclear palsies are due to a vascular lesion (apoplexy), to tumor or abscess inflammation of the cortical cells (encephalitis) or a patch of sclerosis. Nuclear lesions are usually due to a degeneration, being usually a part of the so-called glossolabiolaryngeal paralysis, and may also accompany other degenerative diseases, as progressive muscular atrophy, amyotrophic lateral sclerosis or tabes. If acute they may be due either to the toxins of infectious disease, to alcoholic or mineral poison (polioencephalitis inferior), the infection of poliomyelitis (polioencephalomyelitis), or to hemorrhage. The intracranial portion is usually affected by the exudation of meningitis, frequently syphilitic; by tumor (cerebellopontine angle), or fracture at the base (posterior fossa). The most common cause of facial paralysis is neuritis involving either the intra-osseous or extracranial portions. The former may be due to exposure to cold, traumatism, extension of inflammation from middle-ear disease, operations on the middle ear, possibly to arteriosclerosis. The latter to injuries to the nerve as the pressure

[1] Bruce, Rev. Neurol. and Psychiat., December, 1908, p. 685.

of forceps during labor, implication in cellulitis and growths in the neck and influenza, diphtheria, syphilis, and rheumatism. Disease of the nerve is known as *Bell's palsy*. A few cases have been reported of congenital bilateral facial paralysis.[1]

Symptoms.—In supranuclear or central paralysis the muscles of the lower part of the face only are involved,[2] the patient can close his eye and wrinkle the forehead, and the electrical reactions are normal. If cortical or capsular, there will usually be more or less weakness of the arm and leg upon the same side, and there will be, in addition, involvement of the tongue, and, if a left-sided lesion, aphasia. A lesion in the pons above the nucleus will cause paralysis of the face, arm, and leg upon the opposite side, which will usually be accompanied by some sensory loss, conjugate deviation, and weakness of the ocular muscles. The facial palsy then will be of the supranuclear type. When in the lower part of the pons the nucleus will probably be affected, and as the facial fibers have decussated there will be paralysis of the face upon the same side as the lesion and of the arm and leg of the opposite side. Paralysis of the sixth and conjugate deviation of the eyes may be associated. The facial paralysis will be of the peripheral type (p. 165) (Fig. 7).

FIG. 42

Complete facial palsy. Patient unable to close the eye of the affected side.

Nuclear paralysis is always bilateral. The paralysis in the chronic forms at first is not complete, the orbicularis

[1] Thomas, Jour. Nerv. and Mental Disease, August, 1898, xxv, 571.

[2] During the early stages of a central palsy there may be some weakness of the muscles of the upper part of the face, but it usually soon disappears,

palpebrarum and orbicularis oris often escaping, and develops gradually. It is accompanied by paralysis of other nerves (ninth or tenth) or by symptoms of other diseases. (See Bulbar Palsy, p. 264.) If acute and due to vascular lesion, death soon ensues; otherwise there will be a history of previous infectious disease or exposure to the toxic influences of alcohol or mineral poisons (polioencephalitis). Nuclei of other nerves will probably also be diseased. In *disease of the nerve trunk known as Bell's palsy* the symptoms are most marked and complete (Fig. 42). The eye upon the affected side can be closed but partially or not at all; the lines of the face are smoothed out, especially noticeable in the forehead, and if the patient attempts to frown this side remains motionless. The mouth is drawn more or less to the sound side, and if an attempt is made to smile this is exaggerated. Food collects between the tongue and the cheek. The tears often run over the cheek, owing to the inability to wink. Sensibility is preserved and there is usually no pain. At times there may be slight pain or aching in the region of the ear and mastoid process, and, according to Hunt,[1] if the geniculate ganglion is involved we may have one of the following groups of symptoms:

1. Herpes of the auricle, following fever, general malaise, and neuralgic pains in the ear.

2. In addition to the herpes and symptoms above described, there is a peripheral facial paralysis. There may be also herpes in the course of branches of the trigeminal nerve.

3. In addition to the symptoms described in No. 2, there is evidence of involvement of the auditory nerve, shown by tinnitus aurium, loss of hearing, and sometimes symptoms of Ménière's disease (p. 114).

If due to lesion of the intracranial portion, the eighth nerve, owing to its proximity, is usually involved, causing deafness of nerve origin. If the lesion is here the paralysis may be bilateral, and other cranial nerves are also likely to be

[1] Hunt, Herpetic Inflammations of the Geniculate Ganglion, Jour. Nerv. and Mental Disease, February, 1907, p. 73. Archiv. Inter. Med., June 15, 1910, p. 631.

involved, and headache, optic neuritis, and other symptoms of meningitis or tumor are apt to be present (Fig. 101).

In lesion of the interosseous part, the most common type of facial palsy, in addition to the symptoms described above, there is loss of the sense of taste in the anterior part of the tongue upon the diseased side (for method of testing sense of taste, see p. 59), due to involvement of the chorda tympani. Disease limited to the extracranial portion causes paralysis of all the branches of the nerve, but the sense of taste is preserved. Contractures may eventually occur in the paralyzed muscles, causing drawing of the corner of the mouth to the paralyzed side.

Diagnosis.—The diagnosis must be made as to whether the lesion is central (supranuclear), nuclear, or in the nerve trunk (infranuclear). The differences in the symptoms due to lesions in each of these situations have been detailed in the description of the symptoms.

Prognosis.—The prognosis in supranuclear palsy depends upon the nature of the lesion. The facial paralysis occurring as a part of hemiplegia due to apoplexy often recovers. In nuclear lesions the prognosis is bad. When due to a neuritis, excepting when due to a suppurating middle ear, recovery occurs in most cases. A knowledge of the condition of the electrical reactions is of much help in forming a prognosis. When after ten days there is only a quantitative decrease to galvanism or faradism recovery will probably occur in a few weeks or a month. When reactions of degeneration are present the period of time necessary for recovery will depend upon the rapidity with which they have appeared after the onset of the paralysis and the completeness of their development. When the reaction develops rapidly and is complete, the case is apt to last many months. The patient should be warned as to the possibility of contractures occurring, but these may disappear in time.

Treatment.—The treatment in central and nuclear paralysis of the seventh nerve is that of the lesion causing it. When due to a neuritis the cause, if possible, should be removed. Thus, if due to middle-ear disease this must be thoroughly treated. When the onset has been rapid a blister should be

placed over the mastoid process. This should be followed by hot applications over the nerve. In the so-called rheumatic cases in addition to these measures a purge should be given and salicylic acid in some form administered. Electricity in the form of a constant galvanic current may be used in this stage. In the course of a week iodide of potassium should be substituted for the salicylates and the galvanic current used to cause muscular contraction. The applications should be made every other day. Strychnine is also of value at this stage. If contractures develop, bromides internally, the constant galvanic current, the anode over the muscles, and massage may be of help. Cases in which the paralysis does not improve with these measures may be benefited even after years by anastomosing the facial to either the spinal accessory or hypoglossal, the latter being preferable.

Spasms of the Muscles Supplied by the Seventh Nerve.—**Synonyms.**—Care must be taken not to confound spasm of these muscles with those supplied by the fifth and motor nerves of the eye. Facial spasm has various synonyms, as painless tic, histrionic spasm, mimic spasm, and myospasm.[1]

Etiology.—Irritative lesions of any sort, as tumors, abscess, meningitis, hemorrhages, etc., situated in the course of the seventh nerve neurones, from cortex to periphery, may be a cause of such spasm. It may be caused reflexly by trigeminal irritation, as from a decayed tooth. Blepharospasm, or spasm of the orbicularis palpebrarum, may be caused by eye-strain, foreign bodies in the eye, corneal ulcer, and other irritations. Clonic blepharospasm is also known as nictitating spasm. It may be hysterical. General ill-health, anemia, worry, fatigue, cold, exposure, and pregnancy appear, in some cases, to be exciting causes. In a large number of cases no cause can be found, and are probably due to some disorder of the cortical centres of the facial nerve. (See Torticollis.) Tonic spasm, due to secondary contracture, sometimes follows Bell's palsy. Facial spasm is usually met with after middle life, and is more common in women than in men.

[1] This latter term may be applied to a local muscular spasm anywhere.

Symptoms.—The onset is usually gradual, one or two muscles, generally the orbicularis palpebrarum, being first involved. From this the spasm spreads until either others or all the muscles upon one side of the face, supplied by the seventh, are affected. After a long period of time the spasm may become bilateral, although from the first in some cases twitchings of muscles upon the other side may occur. Those that habitually act together, as the corrugator supercilii and orbiculares of the eyelids, are especially apt to. The spasm may be either tonic or clonic, or both may be combined. Motion or excitement will aggravate or may excite an attack. The spasm decreases or ceases during sleep. It is not attended with pain, but there may be a feeling of fatigue and discomfort and it may interfere with talking and other functions of the facial muscles. Electrical irritability of the nerve may be increased.

Diagnosis.—Facial spasm must be distinguished from tic (p. 431).

In determining the cause the following facts must be remembered: The spasm following Bell's palsy is tonic, and there will be a history of preceding paralysis. If it is hysterical, it will be accompanied by other symptoms of hysteria (p. 460). In reflex spasms careful search will show the cause of irritation, and the eyes, throat, and nose, etc., carefully examined. When due to organic lesion other symptoms are likely to be present or will develop. Thus, a facial spasm may be the first symptom of a cortical irritation (Jacksonian epilepsy). Paralysis also usually coexists or develops later, and the general symptoms of such lesions will be present.

Treatment.—This is unsatisfactory. Sources of reflex irritation, if they exist, must be removed. The general health, if necessary, must be improved. Hypodermic injections of atropine often give relief. Gelsemium and conium in ascending doses by the mouth have been much used. Hot applications or counterirritation over the point of emergence of the nerve may be tried. Weir Mitchell has advised freezing by means of the rhigolene or chloride of ethyl spray. Electricity in the form of a constant galvanic

current, the anode over the various muscles and in front of the ear, with the cathode at some remote point, is sometimes of much service. Cutting the facial nerve and anastomosing with the spinal accessory has been done by Cushing and others with good results. Benefit has been claimed from stretching the facial nerve.

Probably the most efficacious treatment for severe spasm is the injection of ♏xv to xx of 75 per cent. alcohol into the nerve at its exit from the stylomastoid foramen. A hypodermic syringe may be used. Facial paralysis results which disappears and the spasm is relieved for some time, but may return.[1]

Neuralgia.—If we accept the view of Hunt[2] that the facial nerve has sensory functions, the geniculate ganglion being the sensory ganglion and the pars intermedia of Wrisberg the sensory root, we can speak of neuralgia of this nerve. Pain in the face is sometimes felt in the acute stage of a Bell's palsy. Clark[3] has described a case of intractable pain limited to the anterior wall of the external meatus and a small portion of skin in front of the ear (the area said to be supplied by the seventh) which was cured by division of the pars intermedia of Wrisberg.

THE EIGHTH NERVE

Paralysis of those fibers which come from the *cochlea* causes loss of hearing. When there is *irritation* of them, subjective sensations, as noises of various sorts, tinnitus, etc., result. Disease of those fibers which come from the *semicircular canals* causes vertigo (p. 112).

The auditory nerve tract may be affected by middle- or internal-ear disease; by lesions, as either meningitis, especially in epidemic cerebrospinal and syphilitic, or tumor at

[1] Patrick, Jour. Nerv. and Ment. Dis., January, 1909.

[2] Hunt, Jour. Nerv. and Ment. Dis., February, 1907, p. 73. This has been denied by Mills, Jour. Nerv. and Ment. Dis., May and June, 1910.

[3] Jour. Nerv. and Ment. Dis., April, 1910, p. 242.

the base of the brain (cerebellopontine angle); by degeneration in locomotor ataxia and other central diseases; by lesions involving either the nuclei in the upper part of the medulla; the tegmentum (which would cause symptoms on the opposite side); the posterior corpora quadrigemina and internal geniculate body, and the temporal lobes (if on the left side, word-deafness results). (See Aphasia.) Cases in which there was the acute development of deafness, vertigo and paralysis of other cranial nerves, especially the seventh, have been attributed by Hegener[1] to a toxic neuritis of the auditory nerve.

In most cases the lesion is either in the nerve or internal ear. Electricity among other methods may be used to determine which (p. 79). Loss of hearing due to disease of the nerve is detected by methods described on page 58.

Tinnitus may sometimes be relieved by the bromides. In a very severe case, in which deafness had occurred, Ballance[2] divided the auditory nerve, with favorable results.

THE NINTH OR GLOSSOPHARYNGEAL NERVE

Anatomy.—This nerve has three functions: motor, common sensibility, and special sensibility, viz., taste.

The *motor functions* of the nerve are doubtful. It probably supplies the upper pharyngeal muscles. The nerve of common sensibility of the upper part of the pharynx and the tympanic cavity is probably the ninth. The nuclei and centres of origin of these portions of the nerve are in common with those of the tenth, and will be described with them. The course of the nerve fibers of taste is somewhat doubtful. Some believe it is as follows: The fibers originate in the cells of the geniculate ganglion situated on the facial nerve (p. 162). The processes of these cells divide, one division running peripherally, forming the chorda tympani

[1] Quoted in Review Neurol. and Psychiat, April, 1908, p. 255.
[2] Lancet, 1908, vol. ii, see also p. 115.

nerve; this joins the lingual branch of the fifth and supplies the anterior two-thirds of the tongue. The other division runs centrally as the intermediary nerve of Wrisberg, which terminates in the sensory nucleus of the glossopharyngeal nerve. Other branches supply the posterior portion with fibers which conduct sensations of taste. Many neurologists, however, believe that the fifth nerve is the nerve of taste, at least for the anterior portion of the tongue, fibers from the chorda tympani being by them believed to go to the Gasserian ganglion by way of the great superficial petrosal nerve.

The fibers which supply the posterior part of the tongue, palatal arches, soft palate, and epiglottis, arise from the cells of the petrous and jugular ganglion, which are situated upon the nerve in the jugular foramen, as other sensory nerves do, by a division of the process, one division passing peripherally to the structures above named, and the other to the sensory nucleus in the medulla. From there the fibers after decussating enter the lateral fillet and pass through the posterior part of the internal capsule. The cortical termination is probably in or near the uncinate region of the temporosphenoidal lobe (p. 299).

Diseases of the Ninth Nerve.—Etiology.—Owing to its close relation with the tenth and accessory portion of the eleventh nerves, lesions rarely affect the ninth nerve alone. It may be implicated by disease within the medulla, either hemorrhage, acute softening, or degeneration (acute and chronic bulbar palsy); or by involvement of the nerve trunk at the base of the brain with meningitis or newgrowth.

Symptoms.—Symptoms produced by disease limited to this nerve would be disordered sense of taste, especially in the posterior third of the tongue (see Anatomy), and some loss of the power of swallowing. The sense may be less acute or lost (ageusia); more acute than normal (hypergeusia); or perverted (parageusia). The methods used to ascertain the existence of lessening or loss of the sense are detailed on page 59. Loss of the sense is usually unilateral. In hypergeusia sapid substances may be almost painfully appreciated.

When parageusia exists substances may be enjoyed that under normal circumstances would be loathed, and *vice versa*. Parageusia may occur as an epileptic aura. Ageusia and parageusia may occur as hysterical symptoms. Anesthesia of the upper part of the pharynx and loss of the pharyngeal reflex (elevation of the soft palate when the pharynx is irritated) are also present. Irritative conditions of the uncinate region has given rise to subjective sensations of taste (taste auræ) and "dreamy states" (p. 437).

THE TENTH OR PNEUMOGASTRIC OR VAGUS NERVE

Anatomy.—The nerve has both motor and sensory functions. The cortical motor centres that govern laryngeal and pharyngeal movements are situated at the base of the precentral convolution. Fibers from these pass downward near the pyramidal tract (anterior to it in the capsule) and after decussating reach the nucleus ambiguus. This is situated in the medulla between the substantia gelatinosa and the posterior accessory olive, just above the nucleus of the facial nerve. Fibers also reach another nucleus (the so-called sensory) situated just behind the hypoglossal nucleus and extending upward to the floor of the fourth ventricle. The axis cylinders from the cells composing these nuclei form the motor portions of the *ninth* and *tenth* nerves. The external origin is from the side of the medulla anterior to the restiform body. Until recently it was thought that the cells of the lower part of the nucleus ambiguus gave origin to the accessory fibers of the eleventh nerve. These fibers were thought to become the recurrent laryngeal nerve, which supplies all of the muscles of the larynx excepting the cricothyroid. Grabower[1] has shown that it is most probable that such is not the case, but that the laryngeal muscles are all supplied by the tenth. The tenth nerve supplies the

[1] Berl. klin. Woch., December 23, 1895, and Deutsch. Zeit. f. Nervenheilkunde, 1896, vol. ix, Nr. 162.

muscles of the pharynx, and the branches going to the heart, lungs, esophagus, and abdomen contain motor fibers.

The sensory fibers probably arise from two ganglia, named the ganglion of the root or superior ganglion, and ganglion of the trunk or inferior ganglion, which are situated upon its trunk, the former within the jugular foramen and the latter lower down upon the trunk of the nerve. From these, fibers enter the medulla anterior to the restiform body and communicate with the nucleus common to both nerves which extends downward into the cervical region of the cord, and is known by various names, viz., the descending root, the slender fasciculus, respiratory fasciculus, and fasciculus rotundus.

The tenth nerve has numerous connections with the sympathetic system and also with the ninth, the eleventh, the twelfth, and first two cervical nerves. Besides being the motor nerve of the muscles of the larynx and most of those of the pharynx, it is the sensory nerve of the larynx and most of the pharynx, and sends sensory fibers to the viscera (liver, kidneys, lungs, heart, etc.). It also contains vasomotor and secretory fibers.

Diseases of the Tenth Nerve.—Etiology.—The nucleus may be diseased either by acute vascular lesions or by degenerative processes. The latter being most commonly observed in glossolabiolaryngeal paralysis (p. 264). The roots of the so-called peripheral origin may be damaged by meningitis or newgrowth, rarely from aneurysm of the vertebral artery. The nerve trunk in the neck may suffer from deep-seated tumors in that region or from traumatisms. The branch known as the recurrent laryngeal may be damaged by aneurysms and tumors in the thorax. The nerve may suffer, in common with other nerves, in multiple neuritis due to diphtheria, alcohol, and septicemia, and is at times degenerated in locomotor ataxia or other degenerative disease. Temporary functional derangements probably occur, due to the absorption of toxic substances from the intestinal tract. The laryngeal and pharyngeal distribution may be affected by cortical lesions in their centres.

Symptoms.—These may be general or limited to the functions of certain branches, and may be due either to paralysis or irritation, or to both combined. Slowing of the heart, vomiting, and spasm of the laryngeal muscles are the chief symptoms of irritation. Rapidity of the heart's action and paralysis of the pharyngeal and laryngeal muscles are the most obvious paralytic symptoms.

The Pharyngeal Branches.—If a paralytic lesion exists, *difficulty in swallowing* would occur, the food refusing to pass into the esophagus. This symptom is only marked in bilateral lesions. *Spasm* would cause temporary inability to swallow. This symptom is of functional origin and usually is due to hysteria (globus hystericus).

The Laryngeal Branches.—The *motor nerves* of the larynx are the superior laryngeal, which supplies the cricothyroid muscle, and the inferior laryngeal, which supplies the rest.

In addition to the causes above enumerated, paralysis of these muscles is frequently due to hysteria and also to weakness from anemia or exhaustion, congestion or inflammation, and tumors of the larynx. In some cases no cause can be made out.

Laryngeal paralysis is manifested by alteration or loss of voice, derangement of the regulation of the entrance of air during respiration, and defects in the movements of the vocal cords.

Of the latter, according to the degree to which movement is impaired, there are three types: (1) Bilateral abductor paralysis, which results from cold or local inflammation, central or nerve disease; (2) unilateral abductor paralysis, due to pressure on the recurrent laryngeal; (3) adductor paralysis, usually hysterical, but may be due to overuse of the voice or local inflammation.

The symptoms of these various forms are given in the following table of Gowers:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor; no dyspnea.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the larynx occurs usually in rachitic children, and in the condition known as spasmodic croup, but occasionally may occur in adults, in which case it is usually hysterical, but may be a symptom of tetany (laryngismus stridulus). The *laryngeal crises* of locomotor ataxia are probably due to spasm of the adductors. The attacks usually occur at night, and, if severe, are accompanied by cyanosis and dyspnea.

Anesthesia of the larynx may be due to hysteria or to organic lesion. When hysterical, reflex action is preserved; when organic it is lost.

The Cardiac Branches.—Irritation of these nerves or their centres produces slowing of the heart's action, often seen in meningitis, cerebral compression, etc. Slow heart may occur in disturbance of the digestive tract due to auto-intoxication. Paralysis of these branches causes the opposite effect, *i. e.*, rapid action, most often seen in diphtheritic neuritis, less frequently from other toxic influences. When the affection is unilateral not much, if any, disturbance will be noted. Disturbance of nutrition of the centres or trunk, as in neuras-

thenia, may cause an irregular action. Sensory disturbances of the branches are thought to cause pseudo-anginal attacks.

Esophageal and Gastric Branches.—Paralytic disturbance of the esophageal branches is rare, and if present would occasion difficulty in swallowing. Spasm, usually due to hysteria, is more common. Care must be taken not to confound this with organic stricture. The administration of ether in hysterical spasm will cause relaxation of the spasm, when a bougie can be passed. Vomiting is due either to reflex stimulation, or to direct irritation of the nerve roots, as in meningitis or tumor. Gastralgia in some instances is thought to be a neuralgia of these branches; in others it may be due to the direct irritation of their peripheral endings. The "gastric crises" of locomotor ataxia are due to irritation of these branches.

Prognosis.—The prognosis is bad in most cases of organic origin, death sooner or later being the result. Functional disturbances, while they do not threaten life, are often most obstinate and annoying.

Diagnosis.—To determine the seat of the lesion, the nature and distribution of the symptoms and associated symptoms must be taken into account. Disease of the main trunk is rare. Nuclear disease is bilateral and associated with disease of the ninth and twelfth nerves. Disease of one recurrent laryngeal usually denotes thoracic disease. A lesion in the neck making pressure on the nerve is usually apparent. Altered functions due to hysteria would be accompanied by other evidences of hysteria.

Treatment.—The treatment is mainly that of the cause, which must be removed when possible. Electricity, preferably faradism, is often beneficial in cases of hysterical paralysis. It may be applied one pole within the larynx or both applied externally, one to each side of the organ. When the paralysis is secondary to inflammation, electricity and strychnine are indicated. Spasm of the larynx in children (p. 489) may be relieved by a warm bath, the administration of an emetic, as ipecac, or a whiff of chloroform.

THE ELEVENTH OR SPINAL ACCESSORY NERVE

Anatomy.—The cerebral origin of the nerve is found in a nucleus in the medulla a short distance below the nucleus ambiguus. Fibers from it join the tenth nerve. The spinal portion arises from the anterior cornua of the upper end of the spinal cord as far down as the sixth or seventh cervical segments. The roots ascend into the cranial cavity, but leave it again and supply the sternocleidomastoid and trapezius muscles. Its functions are motor.

Paralysis of the Eleventh Nerve.—Etiology.—As it leaves the cranium it may be involved in the exudate of basal meningitis. The cells in the gray matter may be the seat of hemorrhage or of an acute inflammation (acute poliomyelitis), or may be the seat of chronic degeneration (progressive muscular atrophy), due to similar causes that produce nuclear disease elsewhere. It may suffer in caries of the cervical vertebra or from tumors outside the skull, and may be the seat of a rheumatic neuritis.

Symptoms.—Paralysis of one sternomastoid causes difficulty in turning the head to the opposite side, though by the action of the non-paralyzed muscle the head may be turned toward the paralyzed side. Only the upper portion of the trapezius is supplied by the eleventh. Paralysis of this portion would be evidenced by slight drooping of the shoulder and impairment of the power of elevating the arm. In bilateral paralysis the head is without support and consequently cannot be held erect.

Diagnosis and Prognosis.—If due to basal lesion, the several symptoms of meningitis would be present, and other nerves would probably be affected. Cervical caries is accompanied by rigidity, tenderness, and pain on jarring. If due to acute nuclear lesion, the symptoms would be those of acute poliomyelitis (p. 257). A chronic nuclear lesion is progressive and other muscles would soon be affected. Hemorrhage would be sudden in onset, probably following injury. If due to a basal meningitis or cervical caries, remedying of these conditions will likely be followed by a return of power. Chronic nuclear lesions are incurable.

Treatment.—The treatment is that of the cause. Electricity, massage, and strychnine should be employed in peripheral cases.

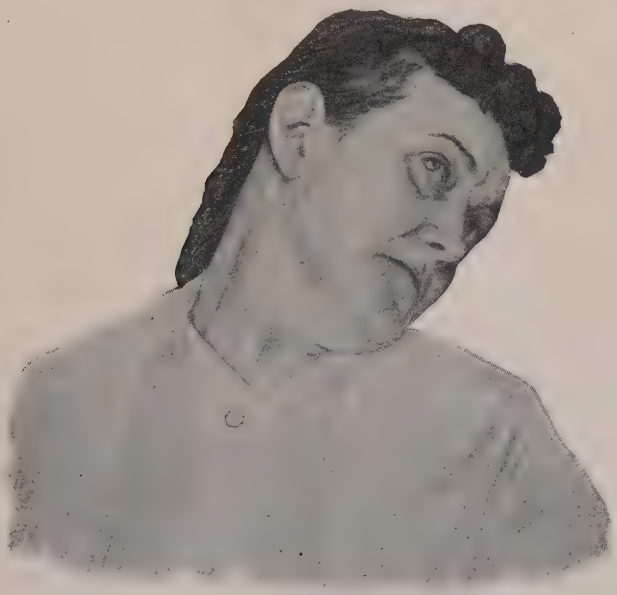
Spasm of the Muscles Supplied by the Eleventh Nerve.—**Etiology.**—Spasm of these muscles—commonly known as *torticollis* or *wryneck*—may be acute or chronic, tonic or clonic. Tonic spasm when acute is usually caused by an inflammation of the muscles and nerve due to cold. This is familiarly known as "stiffneck." The spasm may be due to focal irritative lesions in one of several locations, viz., the spinal cord above the fifth or sixth cervical segments or extramedullary lesion, as newgrowths or hemorrhage, meningitis or bone disease, in the upper part of the vertebral canal; in the medulla; in the trunk of the nerve; in any of the nerves which anastomose with the spinal accessory, and are connected with it in the spinal cord; in the cortical centres. A lesion in one of these situations according to its nature, whether of rapid or slow development, would cause either acute or chronic spasm, which may be either tonic or clonic or combined. In the great majority of cases no cause can be determined and no lesion can be found, but the trouble probably lies in the cortical centres. These cases usually come on in middle life. Those who have a neurotic ancestry are especially predisposed. Overwork, exposure, debility, neurasthenia, reflex disturbances, as bad teeth or eye-strain, appear in some cases to be exciting causes. A number of cases have been observed in weavers. The affection may also be hysterical. It may be a manifestation of tic (p. 430).

Symptoms.—In the *tonic* variety (Fig. 43), when unilateral, the head is rotated so that the chin points upward and toward the opposite side, the occiput being drawn toward the shoulder of the diseased side. When the trapezius alone is affected the head will be drawn backward and the shoulder elevated. Attempts to restore the head to its normal position will cause pain in the affected muscle.

In the *clonic* form the head is thrown into the position above described at more or less frequent intervals. At first the intervals may be long, but as the disease progresses they

become shorter. In severe cases the posterior rotators, which are supplied by the upper cervical nerves, are also involved. These muscles pull the head backward and rotate it *toward* the affected side, which would neutralize the action of the sternomastoid. In most cases, however, the muscles of the opposite side are affected, and hence would exaggerate the spasm produced by the sternomastoid, while the head is

FIG. 43



Spasmodic torticollis.

pulled back more than in pure spinal accessory spasm. The face, shoulder, arm, and other muscles may also take part in the spasm. It is more aggravated during movements of other muscles and usually disappears during sleep. The affected muscles become hard and prominent during the seizure. Bilateral spasm is usually confined to the sternomastoid. By it the head is pulled forward and downward,

the chin pointing toward the sternum. Electrical reactions are normal or increased. In time the muscles hypertrophy. Young children are sometimes met with who paroxysmally nod the head for a minute or more several times during the day (eclampsia nutans). This is thought to be due to a bilateral clonic sternomastoid spasm, probably in most cases due to rickets.

Diagnosis.—Sternomastoid spasm may be confounded with spasm of the *splenius capitis* of the opposite side. In this the head is drawn backward toward the affected side, while the chin is depressed and directed toward the corresponding shoulder. The muscle can be felt as a hard roll beneath the anterior border of the trapezius (Erb). In spasm of the *obliquus capitis inferior* the head is rotated without elevation or depression of the chin. Spasm of these muscles may, however, be associated with spinal accessory disease. Spasm due to organic disease in one of the situations mentioned under etiology would be accompanied by other symptoms of such disease. Tumor or vertebral disease, with meningitis in the cervical region, is probably the most common causes of such spasm. The spasm may rarely be an hysterical phenomenon, but the stigmata of that disease would probably be also present. To distinguish from tic (see p. 431).

Prognosis.—Spinal accessory spasm is one of the most stubborn affections that we have to treat. Relief may be obtained, but can never be promised. The longer the trouble has lasted the worse the chances for relief by medical measures. As regards life the prognosis is good, excepting when due to organic disease, which itself is dangerous to life.

Treatment.—In cases due to organic disease, treatment must be directed to the cause. In the so-called functional spasm sources of reflex irritation must be looked for and removed if found. Benefit may be derived from prolonged and absolute rest, with the head on a low pillow; the bed must be left for no purpose whatsoever, and the patient should be fed by the nurse. Tincture of gelsemium in large doses (gtt. xv, three or four times daily) and atropine in increasing doses, preferably hypodermically, into the affected

muscles once daily, may give some relief. Massage is of benefit, especially when used in connection with rest; also the constant galvanic current, the anode over the affected muscles and the cathode at a remote point, is worth a trial. Counterirritation by means of the actual cautery has been recommended.

Exercises which educate the patient to perform movements either over which he has lost control or which antagonize the abnormal movement (p. 432) are often useful. A slight support to the head acting by suggestion may also relieve the spasm.

When medical means have been given a fair trial and no relief has been obtained, resection of the spinal accessory and posterior branches of the upper four cervical nerves, and even of the muscles themselves, may relieve, but seldom cures.

THE TWELFTH OR HYPOGLOSSAL NERVE

Anatomy.—The cortical centre for the twelfth nerve is situated in the extreme lower part of the precentral convolution. From it fibers run through the internal capsule in close relation with those of the pyramidal tract, and, finally, reach their nuclei, the cells of which begin in the lower part of the medulla, near the middle line of the floor of the fourth ventricle and extend upward as far as the striæ acusticæ. The axones of these cells form the hypoglossal nerve, which is the motor nerve of the tongue, supplying all muscles of the tongue excepting the palatoglossus and pharyngeoglossus. This nerve has connections with the pneumogastric, lingual, upper three cervical nerves, and sympathetic.

Paralysis of the Twelfth Nerve.—Etiology.—Paralysis of the functions of the twelfth nerve may be due to a lesion situated anywhere between the cortex and its peripheral termination, *i. e.*, either a supranuclear, nuclear, or infranuclear lesion. Supranuclear disease is the most common, and may be caused by hemorrhage, softening, tumor, or abscess. Nuclear lesions may be acute or chronic, and are due to the same causes that produce nuclear lesions else-

where (bulbar palsies). They are practically always associated with disease of the nuclei of the ninth and tenth nerves. Infranuclear disease may be caused by softening or tumor in the medulla, by meningitis, fracture or tumor at the base of the brain, by penetrating wounds or tumors in the neck, and vertebral disease.

Symptoms.—The principal symptom is impairment of the power of moving the tongue. If unilateral, the base while at rest is higher on the paralyzed than on the sound side. When the tongue is protruded it deviates toward the paralyzed side, being pushed there by the geniohyoglossus of the normal side. Mastication and deglutition are interfered with.

In bilateral disease the tongue can either not be protruded at all or else but slightly, and then cannot be kept so long. No atrophy occurs in supranuclear lesion; but in nuclear and infranuclear disease it does, the tongue presenting a wasted and wrinkled appearance, and fibrillary tremors are present (Fig. 64). Common sensibility and taste are not interfered with.

Diagnosis.—There should be no difficulty in recognizing that paralysis exists. Supranuclear lesions are usually associated with hemiplegia. Nuclear disease is practically always bilateral and is associated with weakness of the lips, pharynx, larynx, and possibly other muscles (bulbar palsy, p. 264). Crossed paralysis of the tongue on one side and the arm and leg on the other is probably due to an involvement of the root fibers in the medulla. Unilateral paralysis associated with that of neighboring cranial nerves (ninth, tenth) suggests disease at the base.

Prognosis and Treatment.—When a supranuclear palsy is due to hemorrhage or softening (apoplexy) the tongue often recovers power. When due to the other causes of such paralysis the condition is permanent. In nuclear disease the prognosis is bad. If the paralysis is the result of an infranuclear lesion (if the lesion can be removed, as a syphilitic meningitis) the outlook for recovery is fair, otherwise it is bad. The *treatment* is that of the morbid process causing it—*i. e.*, the treatment of meningitis, syphilis, apoplexy, bulbar palsy, or neuritis.

CHAPTER VIII

DISEASES OF THE SPINAL NERVES

THE CERVICAL NERVES

Cervico-occipito Neuralgia. — Etiology and Symptoms.— Neuralgia of the first four cervical nerves, especially the great occipital branch, is produced by the general causes of neuralgias elsewhere (p. 108). It should be remembered that pain in the distribution of these nerves may be due to caries of the cervical vertebræ. It is often associated with neuralgia of the fifth and with torticollis.

The pain is felt in the occipital region and sometimes in the posterior parietal region in addition. It is often bilateral. The scalp may be tender to pressure. Tender points are found: (1) At the angle of the great occipital between the mastoid and the spine; (2) in the triangle between the trapezius and sternomastoid occupied by the cervical nerves; (3) above the parietal eminence.

Prognosis and Treatment.—Excepting when the disease comes on late in life and is rebellious to treatment the prognosis is good.

Counterirritation to the neck and occiput by means of the actual cautery or fly-blister is of great service. Otherwise the medical treatment is the same as that detailed for neuralgia of the fifth nerve.

Paralysis of the Phrenic Nerve.—Etiology and Symptoms. —The phrenic nerve is derived from the third, fourth, and fifth cervical nerves, which emerge from the cord on a level with the second, third, and fourth cervical vertebræ. Paralysis of the nerve is usually due to disease of the cord or its membranes, or of the vertebræ. It is occasionally injured in wounds of the neck, and may be compressed by tumors, aneurysms, etc., in its course through the thorax. Paralysis due to neuritis may follow exposure to cold, and may be

associated with multiple neuritis due to diphtheria or beriberi. Paralysis of both nerves causes inaction of the diaphragm, manifested by the upper part of the abdomen not advancing (even being retracted) during inspiration. On exertion there will be dyspnea and weakness of the voice. Bronchitis and pneumonia are more apt to occur, and are more dangerous when paralysis of these nerves exists. Unilateral paralysis does not cause much inconvenience, and is difficult to discover.

Diagnosis.—It must be remembered that hysterical individuals may breathe for a time without using the diaphragm. Inaction of the diaphragm may be present in peritonitis and diaphragmatic pleurisy, owing to pain caused by such movement. Degeneration of the muscle itself may cause decrease in its power. A bilateral paralysis is usually due to disease of the cord, membranes, or vertebræ, or to multiple neuritis. When due to disease of the cord or membranes paralyses of other parts below the seat of the lesion would be present. In bone disease there would be rigidity of the neck muscles and tenderness to pressure or jarring. Unilateral paralysis is due to either traumatism, neuritis from exposure, compression by tumors, or aneurysm in the neck or thorax.

The **prognosis** depends upon the cause. Bilateral palsy is often fatal. The **treatment** also is that of the cause.

THE BRACHIAL PLEXUS

Paralysis of the Posterior Thoracic Nerve.—Etiology.—The posterior thoracic nerve arises from the fifth and sixth cervical roots, and sometimes from the seventh also. It supplies the serratus magnus muscle. (See Plate I.)

The causes of paralysis of its functions are usually either neuritis due to contusion or a blow on the neck, pressure from a heavy weight carried on the shoulder, violent muscular effort, as raising a heavy weight, exposure to cold, or possibly a toxemia.[1]

[1] A case has been reported of uncomplicated paralysis of the serratus magnus following influenza. Münchener medicin. Wochenschrift, September 6, 1898; quoted in Philadelphia Med. Jour., October 5, 1898.

FIG. 44

Paralysis of the serratus magnus muscle, causing displacement of the scapula when the arm is held forward. (Icon. de la Salpêtrière.)

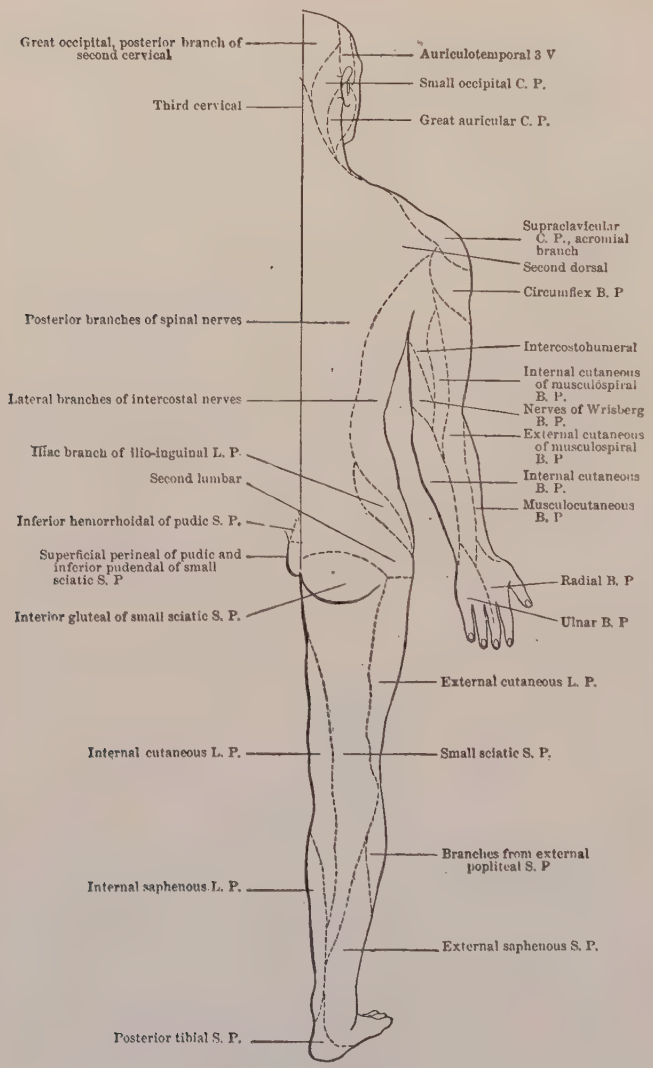
FIG. 45

Paralysis of the serratus magnus muscle, causing displacement of the scapula when the arm is held forward.

FIG. 46

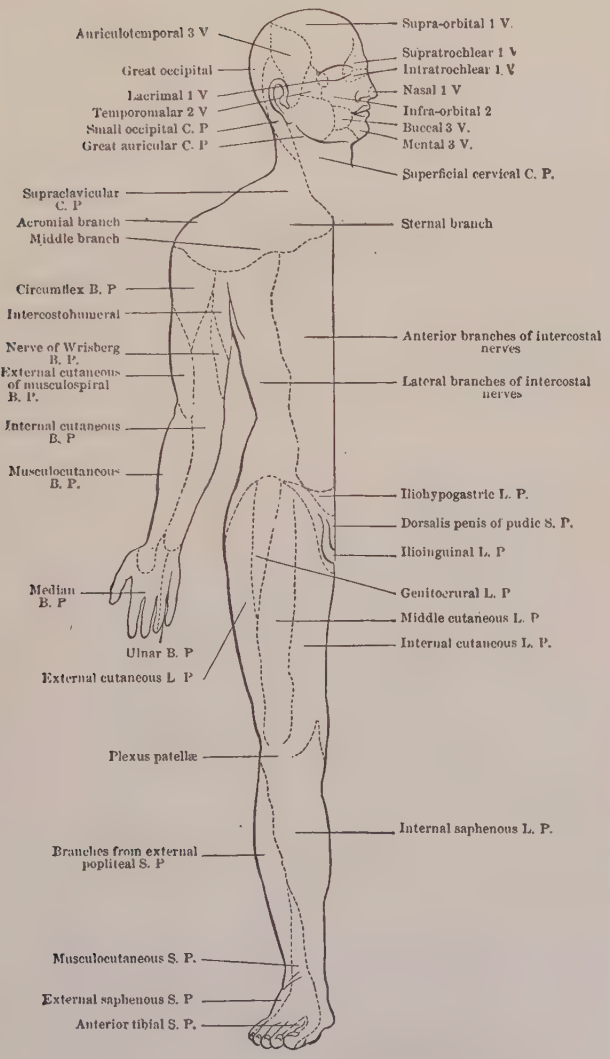
Paralysis of the serratus magnus from injury of the posterior thoracic nerve. Position of the scapula when the arm is abducted. Further elevation of the arm is impossible, as rotation of the scapula is impossible.

FIG. 47


Great occipital, posterior branch of second cervical
Third cervical
Auriculotemporal 3 V
Small occipital C. P.
Great auricular C. P.
Supraclavicular C. P., acromial branch
Second dorsal
Circumflex B. P
Posterior branches of spinal nerves
Intercostohumeral
Internal cutaneous of musculospiral B. P.
Lateral branches of intercostal nerves
Nerves of Wrisberg B. P.
External cutaneous of musculospiral B. P
Iliac branch of ilio-inguinal L. P.
Second lumbar
Internal cutaneous B. P.
Musculocutaneous B. P
Inferior hemorrhoidal of pudic S. P.
Superficial perineal of pudic and inferior pudendal of small sciatic S. P
Radial B. P
Interior gluteal of small sciatic S. P.
Ulnar B. P
External cutaneous L. P.
Internal cutaneous L. P.
Small sciatic S. P.
Branches from external popliteal S. P
Internal saphenous L. P.
External saphenous S. P.
Posterior tibial S. P.

Cutaneous distribution of nerves. (After Flower.)

FIG. 48



Cutaneous distribution of nerves. (After Flower.).

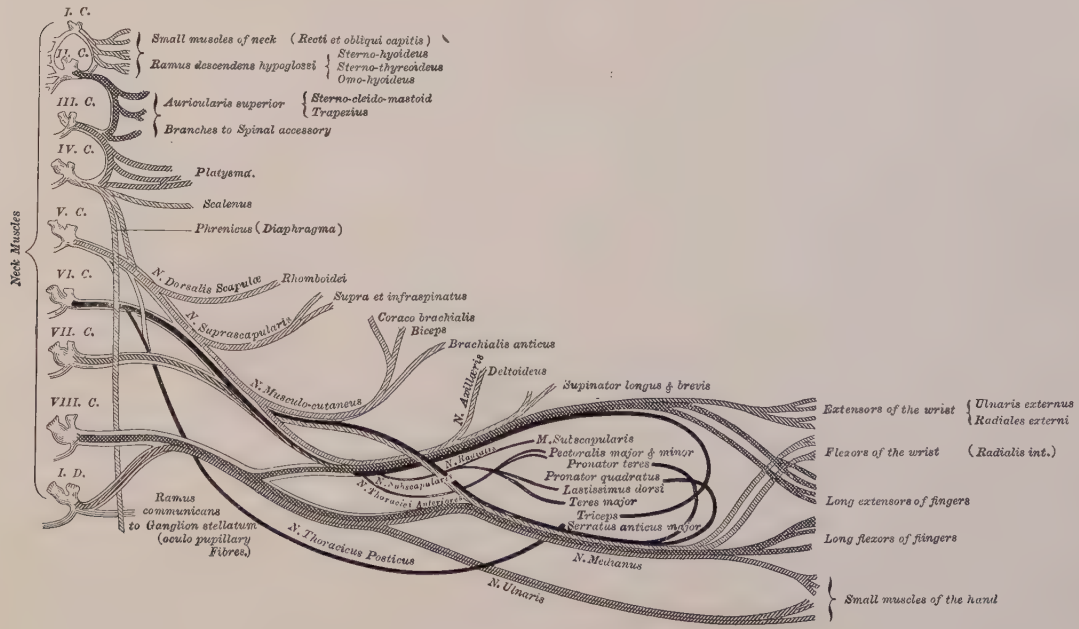
Symptoms.—The symptoms of paralysis of the serratus magnus are: (1) Rotation of the scapula upon its vertical axis when the arm is put forward, with recession of the edge of the scapula from the thorax; the scapula then resembles a wing, and has been termed the "winged scapula"; (2) rotation inward and upward of the lower angle of the scapula when the arm is advanced; (3) weakening or loss of the power of elevating the arm above the shoulder (Figs. 44, 45, and 46). The paralysis is usually unilateral and may be accompanied by pain in the neck and shoulder. An uncomplicated palsy of this nerve is rare.

Paralysis of the Suprascapular Nerve.—Etiology and Symptoms.—This nerve also arises from the fifth and sixth cervical roots (Plate I). It supplies the supraspinatus and infraspinatus muscles. Isolated paralysis of the muscles is rare, but is frequently found associated with paralysis of the deltoid due to injury to the nerve caused by dislocation of the shoulder-joint. Paralysis of these muscles causes loss of the power of outward rotation of the humerus, which is manifested in loss of the power to carry the hand from left to right in writing.

Paralysis of the Circumflex Nerve.—Etiology and Symptoms.—The circumflex nerve arises from the posterior cord of the brachial plexus, which is formed by the fifth, sixth, and seventh cervical roots (Plate I). It supplies the deltoid and teres minor muscles. The nerve is usually injured by falls or blows upon the shoulder or by dislocation of the joint. It may be the seat of a rheumatic neuritis.

In paralysis of this nerve there is loss of the power of raising the arm into or above the horizontal position. Wasting of the deltoid occurs, causing alteration in the appearance of the shoulder; if extreme, there may be a depression between the acromion process and the head of the humerus. The electrical reactions are altered in degree according to the extent of the damage. Trophic changes may occur in the joint and adhesions may form. Care must be taken not to confound loss of motion from paralysis of the deltoid with ankylosis of the joint due to an arthritis. In the latter case there would likely be pain increased by motion, and

PLATE I

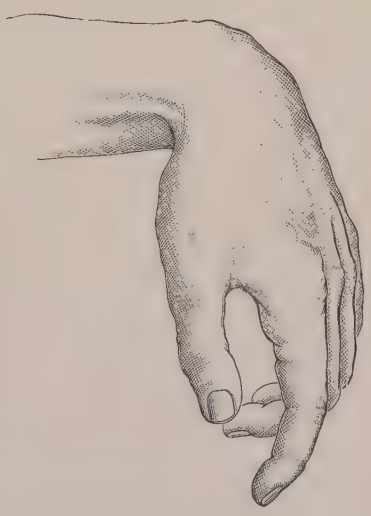


The Cervicobrachial Plexus and its Branches. (Kocher.)

passive movement of the arm would move the scapula also. In some cases there is *loss of sensation* in the upper part of the arm over the muscles (Figs. 47 and 48).

Paralysis of the Musculocutaneous Nerve.—Etiology and Symptoms.—This nerve arises from the outer cord of the brachial plexus, which is formed by a union of the fifth and sixth cervical roots. It supplies the biceps and brachialis anticus muscles (Plate I). Paralysis of these muscles causes loss of the power of flexing the forearm upon the arm, most marked when the forearm is supinated and the supinator longus cannot act as a flexor. There may or may not be *anesthesia*. When present it is found on the radial half of the forearm in front and behind (Figs. 47 and 48). The nerve is rarely paralyzed by itself.

Fig. 49



Wrist-drop in musculospiral paralysis. (Leube.)

Paralysis of the Musculospiral Nerve.—Etiology and Symptoms.—The musculospiral nerve is derived from the posterior cord of the brachial plexus. It supplies the triceps, anconeus, supinator longus, and extensor carpi radialis longior, and by means of its posterior interosseous branch all the extensor muscles of the radial and posterior brachial regions (Plate I).

Paralysis of this nerve is common; it is frequently injured by pressure. (See Pressure Palsy.) It may be torn in fractures of the humerus, or pressed upon by callus, or divided by incised wounds. It is also the cause of the paralysis which sometimes occurs after anesthesia, and is then due to overelevation of the arm. It is frequently involved with other nerves in multiple neuritis.

In paralysis of the nerve high up in the region of the plexus extension of the forearm upon the arm is impossible; the hand cannot be extended upon the forearm and drops when the arm is held in a horizontal position (Fig. 49). The fingers are flexed at their distal points, and owing to loss of the resistance of the extensors the grip is weakened (p. 43). Supination is impaired. If long continued, the excessive flexion of the carpus leads to undue prominence of the carpal bones and the synovial sacs at the back of the wrist. If the nerve is affected low down or the posterior interosseous branch is alone affected, the triceps and supinator longus escape.

Atrophy occurs and the muscles present various degrees of changed electrical reaction.

Loss of sensation, when it occurs, is principally in the distribution of the radial branch and varies in location. According to Head and Sherren[1] it is on the dorsal surface of the thumb, the dorsum of the hand corresponding to the index and middle fingers and the radial side of the forearm. Some observers believe that the dorsum of the index and middle fingers may be involved (Figs. 47 and 48).

Paralysis of the Median Nerve.—Etiology and Symptoms.—The median nerve arises from the outer and inner cords of the brachial plexus. The latter is formed by the eighth cervical and first dorsal roots. It supplies the flexors of the fingers (excepting the ulnar half of the deep flexor), the pronators, the flexor carpi radialis, the two outer lumbricals, and all the muscles of the ball of the thumb, abductor pollicis and the outer half of the flexor brevis pollicis (Plate I). The median nerve is frequently injured or divided by wounds or fractures of the forearm, and injured by blows. It has been rarely injured by excessive action of the pronator radii teres, and possibly may be the seat of a rheumatic neuritis. Paralysis of all the muscles supplied by this nerve, which would occur when the lesion is high up, would cause diminution of the power of flexing the wrist and pronating the forearm. The thumb cannot be brought in contact

[1] Brain, 1905, p. 176 et seq.

with the tips of the fingers. There is inability to flex the second phalanges upon the first, and in the index and middle fingers there is loss of flexion of the third phalanges.

If the lesion is low down, just above the wrist, loss of power in the thumb and fingers, as described above, would only be present unless an ascending neuritis occurred.

Hunt[1] has described cases in which there was paralysis of the thenar muscles with atrophy and without sensory symptoms due to pressure on the thenar branch of the nerve (occupation neuritis). Volkman's contracture may be mistaken for disease of the median nerve (p. 122).

FIG. 50

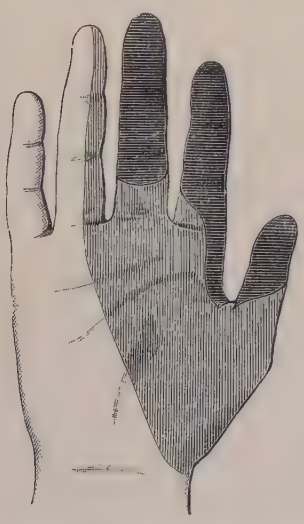
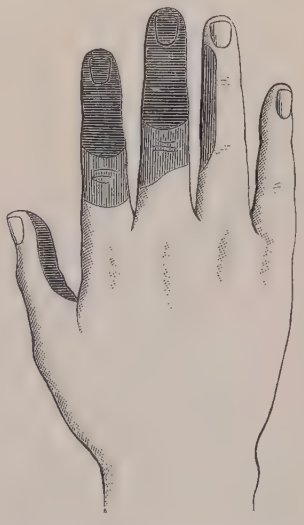


FIG. 51



Showing areas of sensory loss in injuries of the median nerve. Horizontal lines show total anesthesia. Vertical lines show partial anesthesia. (Bowlby.)

Loss of sensation, if present, is limited to the radial side of the palm and the palmar surfaces of the thumb, index and middle fingers, and the radial side of the ring finger. On the dorsal surface loss of sensation occurs in the index

[1] Amer. Jour. Med. Sci., February, 1911, p. 224.

and middle fingers, and on the radial side of the ring finger for a variable distance (Figs. 50 and 51).

Paralysis of the Ulnar Nerve.—Etiology and Symptoms.—The ulnar nerve arises from the inner cord of the brachial plexus. It supplies the ulnar half of the flexor profundus digitorum; the flexor carpi ulnaris; all the muscles of the little finger; all the interossei; the two ulnar lumbricales; the adductor pollicis; and the inner head of the flexor brevis pollicis (Plate I).

The nerve is frequently injured by wounds of the forearm, especially at the wrist, by fractures of the forearm, and at the back of the elbow by pressure or blows. Long-continued flexion of the elbow has caused ulnar paralysis; pressure of a cervical rib and stoop shoulders may also do it (p. 120). There would be, if injured high up, impaired flexion of the hand upon the arm, loss of power of flexing the first or extending the second and third phalanges, of the power of opposing the thumb to the index finger, and of abducting and adducting the fingers. If the paralysis lasts any time, the hand, owing to the action of antagonistic muscles, assumes a characteristic attitude. The hand is slightly overextended and inclines to the radial side of the forearm. Owing to the wasting of the interossei there are depressions between the metacarpal bones, especially marked on the radial side of the index finger on the dorsal surface. The loss of power in the interossei which flex the first phalanges causes their hyperextension, owing to the action of the extensor muscles of those phalanges which are supplied by the musculospiral. For a similar reason there is hyperflexion of the second and third phalanges that are extended by the interossei, owing to the action of their flexors, which are supplied by the median. This deformity is known as the "bird-claw hand" or "claw-like hand" (Figs. 52, 53, and 66). When the lesion is low down, above the wrist, impairment of the functions of the intrinsic muscles of the hand, excepting those supplied by the median nerve alone, occurs. Hunt[1] has described cases in which there was paralysis and atrophy in these

[1] Amer. Jour. Med. Sci., February, 1911, p. 224.

muscles caused by muscular compression of the deep palmar branch of the nerve.

If *anesthesia* occurs it is usually confined to the palmar and dorsal surfaces of the little and ulnar half of the ring fingers and corresponding portion of the hand (Figs. 52 and 53).

Fig. 52

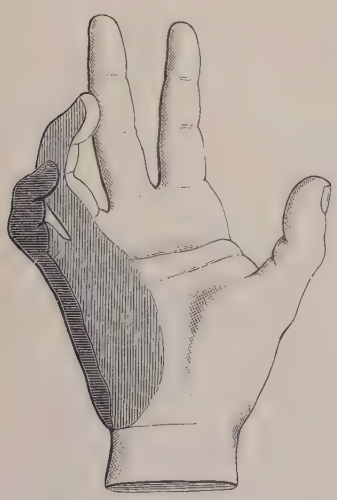
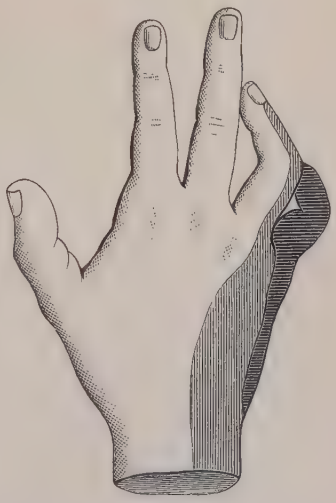


Fig. 53



Showing sensory loss and abnormal position after injuries of the ulnar nerve. (Bowlby.)

Combined Paralysis of the Brachial Plexus.[1]—**Etiology.**—Paralysis caused by involvement of all or nearly all of the branches of the brachial plexus is common. The nerve roots forming the plexus may be damaged within the spinal canal by cervical meningitis or tumor in that region, and by disease of the vertebræ. The causes which concern us here are those occurring external to the spinal canal. These are: (1) Dislocations of the head of the humerus; (2) fractures

[1] See also Paralyses of Upper and Lower Arm Types, pp. 194 and 197.

of the bones of the arm; (3) ascending neuritis; (4) injuries received during birth or later in life; (5) newgrowths situated in the neck; (6) primary brachial neuritis; (7) stoop shoulders (p. 120).

Symptoms.—The symptoms of continued paralysis would, of course, vary according to the severity and distribution of the lesion. When the nerves have been torn or considerably damaged there would be a combination of the motor and sensory symptoms detailed under diseases of the separate nerves. According to the nerves involved, there would be varying degrees of atrophy; De R; trophic and vasomotor disturbances; and if neuritis is the lesion, pain and tenderness. Certain movements are especially liable to be involved —viz., abduction and elevation of the arm (circumflex and posterior thoracic nerves); extension of the hand (musculospiral); flexion of the forearm upon the arm (musculocutaneous). When but slight damage has been sustained the symptoms may consist of nothing more than numbness and slight weakness of the arm, which may disappear in a few days. When due to lesion within the spinal canal irritation symptoms, as shooting pains down the arm and muscular spasms, usually precede the paralysis.

Treatment.—The treatment of paralysis of the muscles supplied by any of the spinal nerves consists in the first place of the treatment of the cause, and next after acute inflammation, if present, has disappeared, of means such as electricity and massage to improve the condition of the muscles. If these measures fail, surgical procedures as either muscle transplantation, nerve anastomosis or the production of arthrodesis may be considered in suitable cases.

Paralysis of the Upper-arm Type (Erb's).—Etiology and Symptoms.—This form probably depends upon injury to the roots of the fifth and sixth cervical nerves at the side of the neck just in front of the edge of the trapezius. The condition is not common, and is met with in infants, and is then often termed "obstetrical paralysis" or birth palsy of Duchenne (Fig. 54). In adults it is usually due to downward pressure on the neck, as by carrying a heavy weight; it may be due to neuritis from other causes.

FIG. 54

1. Typical laceration in brachial birth palsy. Ordinary position in which patient held right arm before operation.
2. Amount of supination of the right hand possible before operation.
3. Showing extent of muscle power in the biceps and deltoid before operation.
4. Showing great reduction in deformity of the right arm nine months after operation. (Compare with 1.)
5. Showing extent of muscle power in the biceps and deltoid nine months after operation. (Compare with 3.)
6. Showing still greater return of muscle power ten months after operation. (Compare with 3 and 5.)

The paralysis involves the deltoid, biceps, brachialis anticus, supinator longus, and supra- and infraspinati. In obstetrical paralysis other muscles are sometimes involved. These muscles are atrophied and present various degrees of De R. Anesthesia in the areas supplied by the circumflex and musculocutaneous nerves may be found. The study of this form of paralysis in infants has recently received a new impetus, owing to the possibility of relieving many of them, who do not respond to other treatment, by operation. Clark, Taylor, and Prout,[1] in an exhaustive study, found that they were due to separation of the head from the shoulders, as by traction on the head when the shoulders are obstructed or on the shoulders in breech presentations. The roots involved are usually the fifth and sixth, although others may be involved so that the entire plexus is affected. The seat of the lesion in most cases is just above or at the junction of the fifth and sixth roots (Plate I). The lesions consist of rupture of the perineural sheath with hemorrhage into its substance, and rupture and laceration of the nerve fibers. There is cicatricial contraction following the organization of the clot and repair of the perineural tear.

Diagnosis.—The diagnosis would depend, in the early stages, upon disuse of the arm by the infant, associated with fretfulness and evidence of pain when the arm is handled, occurring after a difficult labor. The latter symptom, if it persists, indicates a neuritis and is of bad prognostic import. Later in life the diagnosis may have to be made from poliomyelitis, the history, however, should prevent such a mistake being made.

Treatment.—The treatment at first should consist in maintaining and increasing the tone of the muscles with electricity, massage, hot and cold douches, and the prevention of deformities which are liable to occur from overaction of unantagonized muscles. If evidence of neuritis is shown, the limb must be placed at complete rest.

If after a year's treatment recovery is not well under way the roots should be exposed, the diseased area excised and

[1] Amer. Jour. Med. Sci., October, 1905, p. 670.

the nerve endings reunited, an operation which may give good results in a condition otherwise hopeless (Fig. 54). The treatment of this form of palsy in adults should be conducted on the same lines—viz., electricity, massage, etc., and if there is reason to suspect laceration of or pressure upon the nerve roots, operation. (For Prognosis see page 204.)

Brachial Paralysis of the Lower-arm Type (Klumpke).—**Etiology and Symptoms.**—This form is due to involvement of the seventh and eighth cervical and first dorsal roots (Plate I). When the first dorsal root and the communicating branch from the second dorsal are alone involved, a symptom group is produced known as *Klumpke's paralysis.*

Neuritis, either primary or due to pressure from a new-growth in the chest or vertebræ, or a cervical rib[1] is the usual cause. In the first form (seventh, eighth cervical, first dorsal) there is loss of power in the triceps, pronators and flexors of the wrist, the flexors and extensors of the fingers, and the muscles of the hand.

Klumpke's paralysis is characterized by atrophy and paralysis of the intrinsic muscles of the hand and those forming the thenar and hypothenar eminences; anesthesia in the area of ulnar distribution; and ocular symptoms due to involvement of the cervical sympathetic, consisting of myosis on the side of the lesion, sluggish contraction of the pupil, recession of the eyeball, and diminution in size of the palpebral fissure, and loss of ciliospinal reflex.

Diagnosis.—The nerve or nerves affected are determined by a knowledge of the mode of formation, distribution, and functions of the divisions of the brachial plexus already described. Muscular atrophy, loss of reflexes, changes in the electrical reactions (quantitative decrease or De R), show that the lesion is in the peripheral neurone. The presence of pain, tenderness over the nerve trunks, and sensory paralysis would indicate a lesion of the nerve fibers and not of the cells (poliomyelitis). The lesion is usually a neuritis, the cause of which, when not due to traumatism, may at times be difficult to determine. It is then either

[1] Jones, Rev. Neurol. and Psychiat., March, 1908, p. 191.

primary or secondary to some of the pathological conditions detailed under the head of etiology, which must be searched for. Primary neuritis may be confounded with neuralgia. The distinctive points between the two are given on page 110.

If the nerve has been divided, rapid atrophy and complete De R will soon appear. Anesthesia in the distribution of the nerve is also evident.

Involvement of the nerve roots within the canal by disease (tumor, meningitis, vertebral disease) would be distinguished from involvement of the nerves outside the canal by the facts that in the former, in addition to atrophy and paralysis of the muscles of the arm and hand, there would in most cases be a history of muscular spasms preceding the paralysis; in some cases dissociation of sensation, rigidity of the neck muscles, and in the case of vertebral disease pain when the body is jarred, and evidences of impaired conduction in the cord. There would be no tenderness over the nerve trunks, as in neuritis.

Cervicobrachial and Brachial Neuralgia.—Etiology and Symptoms.—This term is applied to pain in the distribution of the four lower cervical and first dorsal nerves. The same causes that produce neuralgia elsewhere are operative (p. 109). Carious teeth have been given as a cause. Oppenheim[1] says that brachial neuralgia is rarely a pure neuralgia, but that such patients may have a commencing tumor in the cortical arm centres, commencing sarcoma of the humerus, high tabes, cervical hypertrophic pachymeningitis, vascular spasm, referred pains, angina pectoris, gallstones, vertebral caries, an occupation neurosis, or, most common of all, neuritis. A cervical rib may also cause pain in the arm; the *x*-ray will reveal the presence of this cause (p. 197).

Tender points, if present, are found in the axilla, over the deltoid, the level of the elbow, and over the ulnar nerve at the annular ligament of the wrist.

Prognosis.—The prognosis depends upon the fact as to whether the cause is removable or not.

[1] Quoted in Philadelphia Med. Jour., vol. i, p. 993.

Treatment.—Complete rest of the arm is important. Intradural division of the posterior roots has been done with success in some cases for the relief of intractable pain in this region, and also in the lumbar.[1] It must be remembered, however, that serious results have also followed this operation and much depends upon the proper selection of the case and the surgical technique employed.[2] Otherwise the treatment is the same as that employed for neuralgia elsewhere. (See Neuralgia of the Fifth Nerve.)

DISEASES OF THE DORSAL NERVES

Intercostal Neuralgia.—Etiology and Symptoms.—Neuralgia of the intercostal nerves is usually caused by exposure to cold or to traumatism, and occurs spontaneously in debilitated individuals.

It is characterized by sharp, stabbing pains shooting along one or more intercostal spaces, with a constant ache in the same situation during the intervals. Tender points may be found at the points of emergence of the nerves from the vertebræ, in the axillary line, and at the sternocostal junction.

Herpes zoster or *shingles* is an affection characterized by neuralgic pains in the course of nerves, most frequently of the dorsal nerves, and the formation of groups of vesicles (herpes) along their course (p. 232).

Mammary neuralgia (mastodynia) is a form of intercostal neuralgia occurring in anemic, debilitated women, and is characterized by neuralgic pains in the region of the mammary gland.

Diagnosis.—Pain in the course of the intercostal nerves may be due to cord disease (meningomyelitis, tumor, tabes) or to vertebral disease. In these cases the characteristic symptoms of these affections would soon develop. Intercostal neuralgia is distinguished from rheumatism of the

[1] Jacoby, New York Med. Jour., August 3, 1907, p. 192, and Jones, Jour. Amer. Med. Assoc., October 7, 1911, p. 1175.

[2] Jour. Nerv. and Ment. Dis., September, 1907, p. 589.

intercostal muscles by the fact that in the latter the sharp, shooting exacerbations do not occur and the pain is intensified by breathing. The acute stage of pleurisy or pneumonia might also be mistaken for it; but in these diseases the pain would be much intensified by respiratory movements and by lying upon the affected side, and there would be present the physical signs of the disorder.

Prognosis.—Intercostal neuralgia is often an affection intractable to treatment.

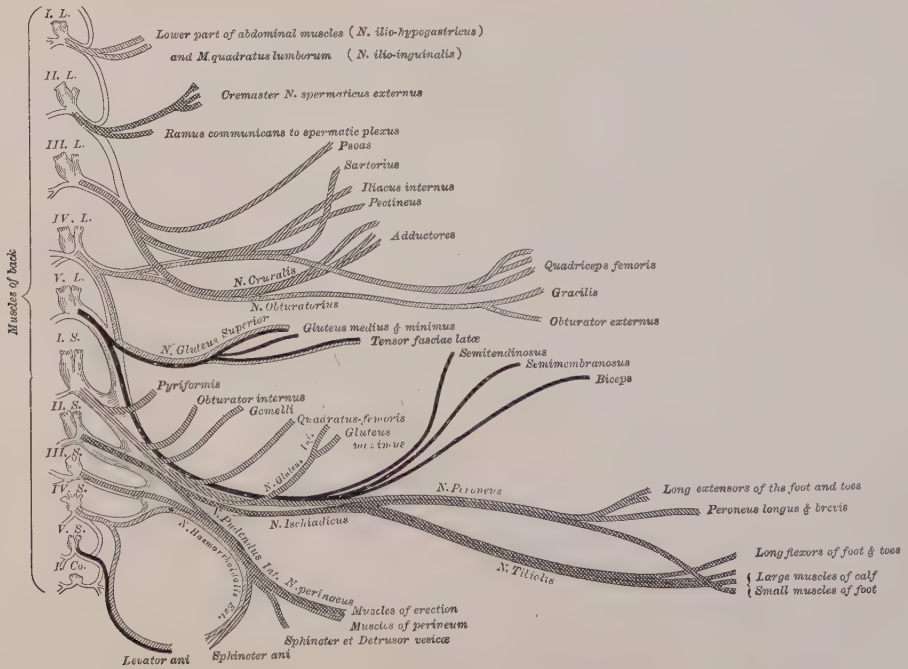
Treatment.—In ordinary neuralgia counterirritation is of benefit. Otherwise the treatment is similar to that employed for neuralgia elsewhere (p. 160).

THE LUMBAR AND SACRAL PLEXUSES

Paralysis of the Nerves Composing the Lumbar Plexus.—**Etiology.**—The lumbar plexus is composed of the first three lumbar roots and one-half of the fourth. Interference with its functions may be due to the pressure of abdominal tumors (ovarian tumors, tuberculosis of the abdominal lymphatic glands), psoas abscess, ascending neuritis, and rarely a primary neuritis. The obturator and anterior crural have been injured during parturition, by dislocation of the hip, and by wounds or blows in the groin. Disease of the vertebræ, meningitis, and tumors springing from the meninges and meningeal hemorrhage may involve the roots composing the plexus. (See Plate II.)

Symptoms.—The branches of the plexus are the iliohypogastric and ilio-inguinal, arising from the first lumbar root; the genitocrural from the first and second and the external cutaneous from the second and third lumbar roots; the obturator and anterior crural from the third and fourth roots. The skin of the lower part of the abdomen, upper and inner part of the thigh, scrotum in the male, labium in the female, are supplied by the iliohypogastric and ilio-inguinal. The genitocrural supplies the skin of the anterior and upper part of the thigh to midway between the pelvis and knee It also supplies with motor fibers the cremaster

PLATE II



The Lumbosacral Plexus and its Branches. (Kocher.)

muscle. The skin of the anterior and outer portions of the thigh as far as the knee is supplied by the external cutaneous. The middle cutaneous branch of the anterior crural supplies the anterior part of the thigh excepting the upper part which is supplied by the genitocrural; the internal cutaneous supplies the skin of the inside of the thigh and the long or internal saphenous that of the inside of the leg from the knee to the base of the great toe. These are also branches of the anterior crural.

Paralysis of these branches would cause anesthesia in the respective areas supplied by them, and in the case of the genitocrural loss of the cremaster reflex (Figs. 47 and 48).

The *obturator nerve* supplies the adductors of the thigh. Its paralysis would cause loss of that function, evidenced by the fact that the affected leg cannot be put across the other. It usually suffers in company with the anterior crural.

The *anterior crural nerve* supplies the iliacus, pectineus, and all the muscles of the front of the thigh excepting the tensor vaginæ femoris. Paralysis of this nerve causes loss of the knee-jerk and more or less marked anesthesia in the area supplied by it. The motor paralysis varies according to the seat of the lesion.

When damaged within the pelvis there would be loss of the power of flexing the thigh upon the body (iliacus) and of extending the leg upon the thigh. When the lesion is lower down only the power of extension of the leg is lost. Muscular atrophy and change in the electrical reactions occur (p. 77).

The *superior gluteal nerve* arises from the lumbosacral cord, which is formed by a union of the fifth with a branch of the fourth lumbar roots. Its isolated paralysis is rare. Abduction and circumduction of the thigh are controlled by this nerve.

Paralysis of the Nerves Composing the Sacral Plexus.—**Etiology.**—The sacral plexus is composed of the lumbosacral cord and the anterior divisions of the three upper and part of the fourth sacral nerves (Plate II). The plexus may be damaged by intrapelvic growths, pelvic inflammations, by vertebral disease, either inflammation or relaxation of the sacro-iliac joint, and within the spinal canal by tumor,

meningitis, or meningeal hemorrhage; by compression during parturition; by ascending neuritis. One of its branches, the sciatic nerve, is liable to suffer outside the pelvis from neuritis (sciatica), and to be injured by traumatism, bone disease, dislocation of the hip, and adjacent morbid growths. The external popliteal (branch of the sciatic) is also liable to suffer from various forms of traumatism, from pressure, and from neuritis. The posterior tibial branch may be damaged by fractures of the leg.

Symptoms.—*Paralysis of the small sciatic* causes loss of power in the gluteus maximus, evidenced by interference with rising from the sitting position. There may also be anesthesia in the perineum and middle third of the posterior part of the thigh and upper half of the calf (Figs. 47 and 48).

The *sciatic nerve* supplies the muscles upon the back part of the thigh. Its branches, the internal and external popliteal, supply all the muscles below the knee (Plate II).

A *lesion near the sciatic notch* would cause loss of the power of flexing the leg upon the thigh and extending the thigh, and paralysis of all the muscles below the knee. When the lesion is below the upper third of the thigh only the muscles below the knee are affected. Anesthesia, if it exist, is found on the outer half of the leg, most of the dorsum, and all of the sole of the foot (Figs. 47 and 48).

The internal popliteal supplies the popliteus, tibialis posticus, flexor longus digitorum, flexor longus pollicis, and the muscles on the sole of the foot. Its paralysis causes loss of the power of extending the foot upon the leg (plantar flexion), and if above the origin of the branch to the popliteus there is loss of inward rotation of the leg when flexed. *Anesthesia* may be found over the outer part and posterior aspect of the lower part of the leg and the sole of the foot (Figs. 47 and 48).

The external popliteal or *peroneal nerve* when paralyzed causes loss of power in the tibialis anticus, extensor longus digitorum, extensor brevis digitorum, extensor proprius hallucis, and peroneal muscles, evidenced by loss of the power of flexing the foot upon the leg, and of extending the first phalanges of the toes (foot-drop). If the weakness lasts

any considerable time, overaction of the sound muscles will cause talipes equinus to develop. If anesthesia is present, it will be found on the outer half of the front of the leg and most of the dorsum of the foot (Figs. 47 and 48).

All the muscles are supplied by the anterior tibial branch excepting the peroneus brevis and longus, which get their supply from the musculocutaneous.

The skin areas supplied by the external or short saphenous, a branch of both internal and external popliteal nerves, and the musculocutaneous and anterior tibial, branches of the external popliteal, and the posterior tibial nerve are shown in Figs. 47 and 48.

The External Plantar Branch of the Internal Popliteal.—Paralysis of this nerve causes loss of power in the muscles of the little toe, the flexor accessorius, the interossei, the two outer lumbricales, and the adductor of the big toe. The lumbricales, interossei, abductor, and flexor minimi digiti flex the first and extend the second and third phalanges of the toes, an action of importance in the propulsion of the body forward in walking. Loss of their action, therefore, is a hindrance to walking, as is also the deformity produced by the action of their opponents causing flexion of the second and third phalanges and extension of the first (similar to the clawhand of ulnar paralysis). Sensory loss, if present, is found in the skin of the outer half of the sole of the little toe and adjacent half of the fourth toe.

The Internal Plantar Branch of the Internal Popliteal.—This nerve supplies the short flexor of the toes, the intrinsic muscles of the big toe except the adductor, and the inner lumbricales. In addition to loss of power of these muscles, anesthesia may be present on the inner part of the sole, the plantar surfaces of the three inner toes, and the adjacent half of the fourth.

Diagnosis.—The diagnosis of diseases of these nerves depends upon the same general principles that have been given for the diagnosis of lesions of the brachial plexus (p. 197). In addition the distinction must be made between a lesion within the spinal canal involving the cauda equina and a lesion outside the canal.

The most common lesion which affects the *cauda equina* is a fracture dislocation of a lumbar vertebræ, causing compression or crushing of the nerve roots. Hemorrhage may be an infrequent cause and also tumor. The symptoms in disease of this region are always bilateral. In extraspinal disease, excepting in multiple neuritis, they would in most cases be unilateral. Both fracture dislocation and hemorrhage would be due to traumatism, and the symptoms would be of sudden onset. If the former, the seat of fracture would be in the vertebræ below the first lumbar. The presence of a neoplasm would be indicated by the slow and progressive development of atrophic paralysis, paralysis of the sphincters, absence of reflexes, and sensory paralysis depending on the nerves involved; intense and increasing sacral pain of a radiating character, and tenderness, often very marked in degree, over the sacrum (Peterson). (See p. 374.)

Prognosis.—The prognosis in paralysis of a spinal nerve depends upon the nature of the cause, the length of time it has been in operation, the amount of damage to nerve fibers that it has inflicted, and the age and general health of the patient. When the cause is pressure exerted upon the nerve, the prognosis is good if that pressure can be removed, even if it has lasted as long as a year. If due to a neuritis the prognosis depends considerably upon its cause; if due to infection or to extension from a suppurating wound, the chances are not so good as if it is caused by traumatism. The longer the cause has been in operation, be it pressure or neuritis, the worse the prognosis. The greater the damage to the nerve fibers, as evidenced by rapid and marked development of the symptoms, the worse the prognosis. The electrical reactions are of assistance in determining this point; the more rapid and typical the development of De R the worse the prognosis. Thus a rapid loss of faradic irritability and the early development of a complete De R show great damage to the nerve fibers, and it will be months before a return of power begins; but if there is a mere quantitative decrease to or no loss of faradic irritability, recovery of power will begin in a few weeks. When the nerve has been divided, the prognosis depends upon the length of time

elapsing before the ends are brought together and sutured. If this is done at once, motion usually returns in two or three months. Partial restoration of function has occurred after a lapse of two years before suturing was performed. Sensibility usually returns sooner than motor power. Its return in cases of nerves that have been divided and sutured is of good omen. The chances of complete and rapid recovery are much better in the young and in those whose general health is good.

Treatment.—The method of treating these cases when due to neuritis has been described on page 122. If due to pressure, it consists in the removal of the cause if possible, and the employment of similar measures to promote the nutrition of the muscles that have been advised for the later stages of neuritis (p. 123).

Neuritis of the Sciatic Nerve (Sciatica).—Etiology.—Sciatica occurs most commonly in middle life or after, in males more than females, and in those whose general nutrition is impaired. It is also more liable to occur in those who are subject to gout or rheumatism, or who have a family history of such. It sometimes follows lumbago. The most common exciting cause appears to be exposure to cold. It seems especially liable to occur in those subjected to sudden and extreme alternations of temperature. Traumatism and violent muscular exertion are also causes. (See also the causes of localized neuritis, p. 120.)

Morbid Anatomy.—The inflammation principally involves the sheath of the nerve, but may extend to the interstitial tissue, and, secondarily, involve the nerve fibers (p. 119). The inflammation is most severe at the sciatic notch and the middle of the thigh. The term "sciatica" is also employed to denote *pain* in the course of the sciatic nerve not due to discoverable organic changes—a true neuralgia.

Symptoms.—The most marked symptom of sciatic neuritis is pain along the course of the nerve, and sometimes along its branches or in its distribution. Its onset may be sudden, when it often seems to be excited by some muscular movement, the pain rapidly becoming very severe. Muscular twitchings are sometimes present during the paroxysms. In

the majority of instances the onset is gradual, dull pain being complained of about the sciatic notch or in the back of the thigh when the leg is moved; especially when moved in a direction that would make tension on the nerve; and by sitting on a hard surface, so that pressure is made on the nerve. This pain becomes more severe and soon spontaneous pain is added, which is usually most severe at the sciatic notch and in the middle of the thigh. It may also extend to the areas in which the nerve is distributed, and may often be felt at the following points: (1) Just above the hip-joint, below the posterior superior iliac spine; (2) in the popliteal space; (3) below the head of the fibula; (4) back of the outer malleolus; (5) on the dorsum of the foot. In character the pain may be burning or dull, associated with exacerbations of shooting pain down the nerve, and is apt to be worse at night. Tenderness when the nerve is pressed upon can usually be found; if not, it can be brought out by stretching the nerve, *i. e.*, causing the patient to lie upon his back, and, with the leg fully extended at the knee-joint, flexing it upon the body (Laseque's sign). In severe cases the patient often stands with the thigh somewhat adducted, the fold of the buttock is depressed and the knee slightly flexed. Owing to the endeavor of the patient to save the leg, the spine is often bent with the convexity to the affected side.

Numbness, tingling, and formication are often complained of. Anesthesia is not found excepting in severe cases. There is often loss of the Achilles jerk. As the disease progresses, loss of power in the muscles may occur, which then become flabby and at times considerably atrophied, with slight electrical changes. If the atrophy is marked, deformities due to the unequal contraction of the muscles may develop. Trophic and vasomotor disturbances may occur, shown by an herpetic eruption and edema of the leg. The neuritis may ascend, involve the lumbar plexus, and cause pain, tenderness muscular weakness in the course and distribution of the anterior crural nerve. In rare instance the inflammation has extended to the spinal cord. With rare exceptions the disease is unilateral.

Diagnosis.—Pain in the course of the sciatic nerve may be due to other causes. Rarely it may be a pure neuralgia. Such cases occur in those who are debilitated and anemic or who have had neuralgias elsewhere. The character of the pain differs, it being spontaneous from the start, not influenced by posture nor aggravated by movement. It is apt to be referred to the branches rather than to the trunk of the nerve. Tenderness along the course of the nerve is absent or very slight. The Achilles jerk is not absent. Intrapelvic disease has caused sciatic pain. In these cases the slight tenderness, which may be present, is out of proportion to the severity of the pain, while a rectal, or if a woman, a vaginal, examination may reveal the cause of the trouble. Hip-joint disease has also been mistaken for sciatica. Careful examination, which should be made in all cases, will always prevent this error. Vertebral disease and tabes dorsalis may cause pain in the course of the sciatic, which, under these circumstances, in the case of the former, would be bilateral; tenderness over the nerve trunk would be absent, and the other symptoms of these disorders would be present. Meningeal thickening, hemorrhage, and tumor involving the cauda equina also cause pain in the sciatic distribution. The pain, however, would usually be bilateral, the sphincters would probably be involved, and anesthesia and muscular paralysis or weakness in the distribution of the different branches of the nerve would be found.

One of the most common conditions to be mistaken for sciatica is disease of the sacro-iliac joint. This may either be an inflammation or what is more common, a strain with relaxation of the joint. It usually follows either a fall, heavy lifting, or twist of the back. A skiagram may clear up the diagnosis. It will be found that the patient gets up from a low chair with the back held stiffly, if he bends sideways from the hips there will be limitation of motion on the affected side, applying Kernig's test (p. 220) will cause pain in the joint and grasping the crests of the ilia and separating them and drawing them together will cause pain.[1]

[1] Amer. Jour. Med. Sci., June, 1911, p. 855.

A therapeutic test may be applied by supporting the joint with broad strips of adhesive plaster drawn firmly from one anterior superior iliac spine to the other, when, if the trouble is in the joint, the pain will be relieved.

Prognosis.—The prognosis of primary sciatica is usually good as regards ultimate recovery. The length of time necessary for this result to be attained depends upon the severity of the symptoms and the ability of the patient to undergo proper treatment. It may last for months or years and relapses sometimes occur.

Treatment.—Rest is of the greatest importance. A successful plan is that advocated by Graeme Hammond: The patient is put to bed and the leg immobilized by a long hip splint. Hot-water bags are kept along the thigh from the sciatic notch to the popliteal space. The constant galvanic current is applied once daily for five minutes, the anode being placed under the hip at the gluteal fold and the cathode at the sole of the foot; both electrodes should be large. For the acute pain at the beginning, large doses of phenacetin, gr. xv, are administered. In mild cases rest in bed need not be insisted upon, but the leg should be kept flexed and crutches used for walking; sitting upon hard or cold surfaces must be avoided. Ice-bags sometimes act well for the relief of pain, as does also the constant galvanic current used, as above described. In cases of long standing, counterirritation by the long-continued use of small blisters, or better by light burnings with the actual cautery, is of service. Hypodermic injections of atropine, distilled water, or cocaine may give relief. The latter must be used cautiously. In cases of rheumatic origin the salicylates are indicated. Nitroglycerin may also at times prove of service. Good results have been reported from the deep perineural injections of salt solution.[1] When all else fails the nerve may be cut down upon and stretched, and if adhesions between the sheath and trunk are present they should be loosened. Stretching the nerve, while it sometimes gives

[1] D'Orsay Hecht, Jour. Amer. Med. Assoc., February 6, 1909, p. 444.

great relief, in others seems to aggravate the pain and weakness.

Constitutional treatment must not be neglected, the system being built up by appropriate tonics, and if a gouty or rheumatic patient, the diet and habits regulated.

Metatarsal Neuralgia or Morton's Disease.—This term has been applied to a condition characterized by severe pain, excited by walking or standing, which usually starts at the base of the fourth toe, soon involves the entire foot, and frequently extends up the leg. It is either throbbing, lancinating, or both combined. Pain is excited by pressure over the head of the third and fourth metatarsal bones and sometimes by pressing the metatarsal bones together. It is caused by a neuritis due to pressure of the metatarsal bones upon the branches of the plantar nerves. Talipes planus, or flat-foot, is frequently associated.

Treatment.—The treatment consists, if flat-foot is present, in the relief of that condition by proper mechanical means (plantar springs, etc.) and strengthening of the muscles of the leg and foot by exercise, massage, and electricity. If these measures fail, excision of the head of the metatarsal bone, where the pain is located, should be performed.

Neuralgia of the Lumbar Plexus.—When neuralgic pains affect the lower half of the trunk they are termed lumbo-abdominal neuralgia. Painful points may be found near the middle of the iliac crest and at the lower part of the rectus muscle. Care must be taken not to confound the affection with the pain due to abdominal or intrapelvic disease and rheumatism of the muscles. (See Treatment of Cervicobrachial Neuralgia, page 199.)

CHAPTER IX

INFLAMMATION OF THE MENINGES

The dura mater is composed of two layers: the inner layer is thin and has a smooth surface covered with endothelium; the outer layer is thicker and serves as a periosteum for the bones. Either of these layers may be affected by inflammation. Inflammation of the dura mater is termed *pachymeningitis*, either external or internal, according to the layer affected.

EXTERNAL CEREBRAL PACHYMENINGITIS

Etiology.—The usual causes are traumatisms of various kinds to the head, sunstroke, caries of the cranial bones due to syphilis, middle-ear disease, ozena, and secondary infection due to inflammation of the surface, as erysipelas of the head or face. Sometimes no cause can be traced.

Morbid Anatomy.—In acute cases there are at first redness and edematous swelling of the tissues, with later the formation of pus, which accumulates between the dura and the bone. In the more chronic forms there is thickening of the membrane, which is more or less firmly attached to the bone and pia arachnoid. Osteophytes may be formed upon the surface of the bones and in the dura (ossifying pachymeningitis). The disease may extend to the pia and cord.

Symptoms.—The symptoms are indefinite, the diagnosis often depending upon the discovery of the cause. They consist of severe headache, vertigo, delirium, and sometimes convulsions; fever may or may not be present. It should be remembered that similar symptoms often precede the infectious diseases. If a collection of pus is formed which

makes pressure upon the motor cortex, there may be more or less pronounced paralysis upon the opposite side.

Prognosis.—The acute form is serious; the chronic variety, while rebellious to treatment, need not necessarily cause death.

Treatment.—At the outset antiphlogistic measures, as purgatives, cold to the head, wet cups at the back of the neck, and ergot, bromides, and mercurials internally, should be employed. If there is reason to suspect fracture or the presence of pus, trephining is indicated. In the chronic forms the use of potassium iodide and mercury, in full doses if syphilis is suspected, is indicated; and vigorous and prolonged counterirritation at the back of the neck, preferably by lightly burning with the actual cautery. If these measures fail, the removal of a button or two of bone with the trephine may give relief.

INTERNAL CEREBRAL PACHYMENINGITIS

Etiology.—The inner layer of the dura mater is much less frequently diseased than the outer. It may be met with in two forms: purulent and hemorrhagic. In the purulent form it may be inflamed in company with the outer layer as a consequence of trauma, or a purulent inflammation of the pia may extend to it.[1] The latter form, known as hemorrhagic internal pachymeningitis, or hematoma of the dura mater, while rare, is more common than the purulent meningitis. This condition is met with chiefly in males, and is a disease of old age. According to Gowers, more than one-half the cases occur in persons over fifty; 25 per cent. of these occurring after seventy. It is most frequently found in cases of chronic insanity and in those who have been the victims of chronic alcoholism. It may occur in cases of profound anemia and diseases of the blood and bloodvessels. It has also been found in rachitic and ill-nourished babies.[2]

[1] Leszynsky has recently reported a case of inflammation (purulent) practically limited to the internal layer, due to middle-ear disease.

[2] See Herter on Hemorrhagic Internal Pachymeningitis in Children, Amer. Jour. Med. Sci., August, 1898, p. 292.

Morbid Anatomy.—The morbid anatomy of the hemorrhagic form presents one of three conditions: (1) A delicate subdural vascular membrane, formed by the penetration of bloodvessels and granulation tissue into an inflammatory exudate; (2) simple subdural hemorrhage; (3) a vascular membrane with the blood clot. Cysts may occur within the membrane. The source of the bleeding is probably the bloodvessels of the dura. Atrophy of the convolutions is usually associated with the hemorrhage.

Symptoms.—The symptoms of either form are indefinite. Those of the purulent form are those of the form of either inflammation of the external layer or pia that it accompanies.

In the hemorrhagic form no symptoms may be noticed, or the patient may have at varying intervals apoplectic attacks, the symptoms of which are of brief duration. These are slowness and irregularity of the pulse, contracted pupils with poor or no response to light, vomiting, and coma. With these there may be more or less complete paralysis of a hemiplegic form. In some cases severe headache, vertical or temporal, may be a prominent symptom.

Diagnosis.—The condition may be expected to exist when symptoms such as have been described occur in alcoholics or cases of chronic insanity, especially terminal or senile dementia or paresis.

The **prognosis** is bad, a fatal result being almost certain to occur in time.

Treatment.—During the attack the head should be elevated, with the application of cold and the administration of purgatives. If possible, the cause, as excessive use of alcohol, should be removed.

INTERNAL SPINAL PACHYMENINGITIS AND PACHYMENINGITIS CERVICALIS HYPERTROPHICA (CHARCOT AND JOFFROY)

Etiology.—This may be associated with external pachymeningitis due to vertebral caries or syphilis. There is, more-

over, a special form which usually involves the cervical region, but may occur rarely in either the medullary, dorsal, or lumbar regions, and is known as hypertrophic cervical pachymeningitis. Alcoholism, exposure, syphilis, and trauma are causes.

Morbid Anatomy.—The morbid anatomy of the latter variety consists of a chronic inflammation of the inner layer of the dura, which produces a layer deposit of fibrous tissue with consequent great thickening of the dura. Hemorrhages may occur within the dura or in the newly formed layers of tissue upon their inner surface. These resemble those that occur in internal cerebral pachymeningitis. The pia finally becomes adherent to the dura, and the newly formed strata-like tissue binds together the membranes, nerve roots, and cord in one mass with resultant compression of the cord and degeneration of its tissue—in other words, a compression myelitis results.

Symptoms.—The first symptom in the usual cervical form is pain of a neuralgic character in the occiput, neck, and shoulders, radiating down the arms. With this may be associated twitchings of the arm muscles. Atrophy and paralysis, beginning in the thenar, hypothenar, and interosseous muscles, soon follow (Figs. 65 and 66). These spread to the muscles of the shoulder and arm. The paralysis is usually flaccid, but there may be rigidity. Fibrillary tremors are present. Stiffness and scoliosis of the cervical spine occur. As the disease progresses, pressure is made on the cord by the thickened membranes and spastic paraplegia, without atrophy and with increased reflexes, and with or without paralysis of the bladder and rectum, makes its appearance. The sensory changes vary in type. The so-called dissociation of sensation may be present in some areas (p. 49). In others there may be tactile anesthesia or merely delayed sensation. They may have a paraplegic or a hemiplegic distribution. Owing to involvement of the cervical sympathetic, pupillary changes may occur (p. 67).

Diagnosis.—The diagnosis must be made from syringomyelia, vertebral caries, tumor, amyotrophic lateral sclerosis, progressive spinal muscular atrophy, and primary brachial

neuritis. When the dissociation of sensation is present, the diagnosis from syringomyelia is difficult; the occurrence of pain, especially of a shooting character, or a history of syphilis would point to pachymeningitis. Meningitis due to vertebral caries would be associated with tenderness upon pressure or jarring the body, and other evidence of bone disease. In tumor the symptoms would probably be more gradual and irregular in their progress, and deformity of the neck would not develop. In amyotrophic lateral sclerosis there is no severe pain, if any, nor anesthesia. In progressive muscular atrophy a similar distribution of atrophy, with knee-jerk absent or not increased, and no sensory changes would occur. Primary brachial neuritis (the radicular form) is not associated with cord symptoms; neither would there be deformity. The atrophy would be unilateral and not so pronounced. It is more apt to occur in women; pachymeningitis is more common in men. If the process occurs in the lumbar region, as it rarely does, there are shooting pains down the legs and absent knee-jerks, resembling somewhat locomotor ataxia, multiple neuritis, and tumor, but differing from the former by the presence of muscular atrophy and paralysis; from multiple neuritis, by the absence of tenderness over the nerve trunks and the presence of evidences of cord involvement; from tumor involving the meninges the diagnosis may be most difficult, the points detailed in the diagnosis of the cervical condition from tumor may aid.

As regards cure, the **prognosis** is bad; but life may be preserved for years.

Treatment.—The treatment consists of rest; counterirritation by the cautery or blister; and iodide of potassium and bichloride of mercury internally. Bed-sores must be guarded against; and if paralysis of the bladder exists, the catheter used and the occurrence of cystitis avoided if possible.

SPINAL MENINGITIS (ACUTE)

Etiology.—Spinal meningitis also occurs in which either the pia-arachnoid alone is involved or the dura is involved with

it. It may be acute or chronic. The former may be due either to tuberculous or malignant disease of the vertebræ. Syphilitic bone disease may also cause it. Tuberculous disease of the cerebral meninges may extend to those of the cord as may other forms of cerebral meningitis. It may follow any of the infectious diseases, and is then usually associated with myelitis; it may be due either to extension of purulent inflammation, septicemia, or follow an injury to the back. In association with cerebral meningitis it is present in the epidemic disease, or as a sequel of the infectious diseases (p. 218).

Morbid Anatomy. — Following a hyperemia of the finer bloodvessels of the meninges, develops a serous, fibrous, and purulent exudate which is deposited in the meshes of the pia and arachnoid and causes the cerebrospinal fiuid to become turbid. The membranes become covered with it and bound together by it. The process usually extends to the cord. (See Myelitis.) If tuberculous in origin miliary tubercles will be found and the exudate is not purulent.

Symptoms.—If cerebral meningitis is present the spinal symptoms may be masked. The onset may be with chill and fever of irregular type, followed by pain and rigidity of the spine especially when moved, shooting pains in the limbs, general hyperesthesia, opisthotonos, spasms of the muscles of the abdomen and limbs, increased reflexes, and Kernig's sign (p. 220). As the disease progresses, paresis or paralysis, incontinence of urine, hypesthesia and hypalgesia, irregularity of heart and respiration may occur. Lumbar puncture may aid in the diagnosis, especially as to the germ causing the inflammation (p. 221).

While meningitis may occur as a sequel of any of the infectious diseases, notably pneumonia, typhoid fever, influenza, and rheumatism, a condition may also occur in the course of these diseases in which the symptoms simulate those of meningitis, but no pathological change in either the cerebrospinal fluid or membranes is found. This has been called either *meningism* or *meningismus*. An examination of the fluid obtained by lumbar puncture will usually clear up the diagnosis, as in meningismus it has all normal characteristics and is sterile.

Prognosis.—Patients frequently die in from a few days to a few weeks. The prognosis is especially bad in tuberculous cases. Septic cases and those following infectious diseases may recover with possibly some paralysis.

Treatment.—Absolute rest in bed, preferably on an air- or water-bed. The patient should be kept on the side as much as possible. Ice-bags may be applied to the spine and blood withdrawn by cups or leeches. Hexamethylenamin in full doses should be employed. In the later stages counter-irritation to the spine is indicated. Laxatives should be given and mercurial inunctions have been recommended. Pain must be relieved by phenacetin or similar drugs, the bromides, and, if necessary, opium. If evidence of pressure on the cord is present, lumbar puncture should be done.

SPINAL MENINGITIS (CHRONIC)

Etiology.—This may follow the acute form. The majority of cases are due either to syphilis, tuberculous or syphilitic disease of the vertebræ, pressure by a tumor or aneurysm, or traumatism. It may also be present in association with chronic diseases of the spinal cord, as tabes, myelitis, etc.

Morbid Anatomy.—In cases which follow the acute or in those which occur in association with chronic cord diseases there is merely a connective-tissue formation, with distention of the vessels, leukocytosis, and thickening. There is also a form in which there is an increase of cerebrospinal fluid which may be either diffuse or circumscribed (p. 216). If syphilitic, gummatous, gelatinous masses surrounding the cord are found associated with thickening of the vessel walls. The process usually extends into the cord, causing areas of degeneration there. If due to tuberculous disease of the vertebræ, caseous material and pus cover and infiltrate the membranes and surround the cord. (See Compression Myelitis.)

Symptoms.—These consist of weakness of the limbs (usually paraplegic), pains in the back and extremities, increased reflexes, either hyperesthesia or diminished sensi-

bility, and disturbances of the sphincters. These either develop gradually or follow the acute process.

Diagnosis.—Traumatic lumbago (p. 480) may be mistaken; in this, however, the evidences of cord disturbance are not great, unless hysteria is associated, when the absence of the Babinski phenomenon and the presence of other phenomena of hysteria will distinguish the two. If any doubt exists an increased number of leukocytes would probably be found in the cerebrospinal fluid obtained by lumbar puncture when meningitis is present (p. 221). Myelitis is usually more or less associated; if it exists without meningitis, there is very little if any pain; and if it is the primary condition, the cord symptoms (p. 381) are more pronounced.

Treatment.—Mercury and iodides in full doses if syphilitic. Counterirritation to the spine, long-continued warm baths, warm spinal douches and measures to improve the general health should be used in all cases. If spinal caries is present appropriate treatment for that condition should be employed.

SEROUS SPINAL MENINGITIS (DIFFUSE AND CIRCUMSCRIBED)

Horsley[1] has described a number of cases in which at operation a large excess of cerebrospinal fluid was found. The cord was shrunken. A similar condition has been found in which either disease of the vertebræ, tuberculous or syphilitic meningitis, syringomyelia or tabes was associated. An idiopathic condition also occurs in which the fluid is circumscribed in the pia arachnoid. When the dura is slit the opening is filled by a more or less opaque pia that bulges forth under tension of the contained fluid which is clear. This has been termed *circumscribed serous spinal meningitis*.[2]

Etiology.—In addition to the conditions above mentioned some cases seem to follow the infectious diseases (influenza),

[1] British Med. Jour., February 27, 1909, p. 513.

[2] Weisenburg, Amer. Jour. Med. Sci., November, 1910, p. 719, and Mills, Jour. Nerv. and Ment. Dis., September, 1910, p. 529.

while the circumscribed type has apparently been caused by trauma.

Symptoms.—These resemble those of tumor of the cord.

Diagnosis.—This is difficult to make from tumor, the condition being found either at operation or autopsy. Pain is usually excessive and Horsley believes it is more diffuse than that caused by a hard tumor. Hyperesthesia is also more pronounced. Weisenburg believes that variation in the motor and sensory symptoms from day to day is indicative of serous meningitis as is also a slow progression of the symptoms.

Treatment.—Operation is the treatment, the cord being exposed at a point indicated by the localizing signs (p. 368). Horsley in addition to evacuating the fluid, irrigated the subdural space with a 1 to 1000 bichloride of mercury solution.

CEREBRAL LEPTOMENINGITIS—VARIETIES

Inflammation of the cerebral pia mater, or **leptomeningitis,** occurs as one of the following types: (1) *Simple*, due to either the pneumococcus, streptococcus, staphylococcus, typhoid bacillus, gonococcus, or colon bacillus, and those of other infectious diseases. The first two are the most common. (2) *Epidemic cerebrospinal*, due to the Diplococcus intracellularis meningitidis. (3) *Tuberculous*, caused by the tubercle bacillus. (4) *Serous meningitis.* (5) *Syphilitic* meningitis. These forms may be acute or chronic, usually the former; the latter, when it occurs, being a sequel of the acute.

ACUTE SIMPLE CEREBRAL MENINGITIS

Etiology.—It may occur as a sequel or accompaniment of septicemia or pyemia, and of any of the infectious diseases. Pneumonia is the disease in which this most frequently occurs. Meningitis may also sporadically be caused by the

microörganism of pneumonia (the Pneumococcus or Diplococcus pneumoniæ of Fraenkel and Weichselbaum) independently of the existence of pneumonia; by diseases of the frontal sinuses and upper nasal passages; or a brain abscess may reach the surface and cause a meningitis. It may be due to disease or injury of the cranial bones, and, what is the most frequent cause of all, infection from disease of the *middle-ear* and *mastoid cells*. Alcoholism, chronic nephritis, arteriosclerosis, gout, and the wasting diseases of children are predisposing causes. The disease occurs most frequently in males and in the young. It may be combined with spinal meningitis (p. 214).

Morbid Anatomy.—The pia arachnoid is either thickened and congested or infiltrated with yellowish purulent exudation, which is more pronounced along the course of the veins. The ependyma may be granular and thickened, and the ventricles dilated and filled with turbid fluid or lymph. The cortex may be softened, and foci of hemorrhage and encephalitis may be found.

Symptoms.—As the inflammation may be confined either to the vertex or the base, or to limited areas in these localities, and also may or may not be attended with the presence of pus, it will be understood that while there will be certain symptoms common to all these forms, there will be special symptoms dependent upon the location and character of the inflammatory process.

In all forms there is usually a short prodromal period, in which the patient suffers from malaise, languor, irritability, headache, loss of appetite, vomiting, and vertigo. This period may be absent, the attack being ushered in suddenly by a chill. These are succeeded by an *irritative stage* in which the symptoms consist of fever; violent and constant headache; delirium; hyperesthesia of the skin and special senses; vomiting, independent of the presence of food in the stomach; contracted and perhaps unequal pupils; rigidity of the muscles of the neck; retraction of the head; and retraction of the abdominal muscles, forming the so-called boat-shaped abdomen. In some cases a general rigidity may be present. A symptom found in

many cases of leptomeningitis is inability of the patient fully to extend the legs when sitting up, while there is ability to do so when recumbent. If such a patient is made to rise from the recumbent to the sitting position and an attempt made to extend the leg on the thigh, there is contraction of the flexors, which prevents the leg being extended. The same phenomenon is seen if while recumbent the thigh is flexed on the abdomen and an attempt is made to extend the leg on the thigh. When the patient is recumbent and the thigh extended the leg can also be extended (Kernig's sign).

Muscular twitchings or convulsions take place. Drawing a dull point along the skin causes a red line to appear (tache cérébrale). The tongue is heavily furred, and there are obstinate constipation and scanty and perhaps albuminous urine. Optic neuritis may or may not occur. The pulse is apt to be irregular, and may be slow—50 to 70. As the disease progresses, the paralytic stage sets in, and muscular paralyses succeed the twitchings and convulsions. The patient becomes stupid, and, finally, comatose, the pupils dilate, the respiration becomes of the Cheyne-Stokes type, the skin becomes clammy, and involuntary evacuations of urine and feces take place. When the disease is limited to the vertex, delirium, headache, and convulsions are more marked than in basal meningitis; and when paralysis occurs it is likely to be that of either an entire limb or of a hemiplegic type. Optic neuritis and rigidity of the neck muscles are not so likely to be present. In meningitis of the base, paralyses of various cranial nerves occur; optic neuritis, rigidity of the neck, occipital headache, and vomiting are liable to be marked. A hemiparesis due to pressure upon the crus may be a symptom also.

Meningitis confined to limited areas may be determined by the headache being limited to a small area of the head, and by focal symptoms, as disturbances of certain motor centres.

When the meningitis is due to purulent infection, chills occur, succeeded by high temperature, and the course is apt to be very rapid.

Diagnosis.—There should usually be no difficulty in determining that meningitis is present when one of the exciting causes mentioned is followed by symptoms such as have been detailed above. Doubtful cases may be decided by lumbar puncture and the nature of the infection, whether due to streptococci, staphylococci, pneumococci, Diplococcus intracellularis (epidemic type), or tubercle bacilli can also be determined by cultures from, and the appearance of the fluid. It is usually turbid and may contain pus, the leukocytes are greatly increased, the polynuclear form being found in all forms but the tuberculous, when the mononuclear predominate. The pressure and the globulin are increased (pp. 96 and 97).

Meningitis of this type may be confounded with abscess, and, as both may be produced by similar causes, they may co-exist. Explosive manifestations after the disease has been for a long time latent (see Brain Abscess) are characteristic of abscess. Unilateral symptoms are more common in abscess; exaggerated deep reflexes upon one side may be found. Subnormal pulse and elevation of temperature point to abscess. The cranial nerves at the base are more apt to be involved in meningitis than in abscess.

Cerebral rheumatism may be mistaken. This occurs in the course of acute articular rheumatism, when in association, with a delirium in which there is marked fear and frightful hallucinations, meningitic symptoms may occur. These are usually not so pronounced as in purulent meningitis. Examination of the cerebrospinal fluid, which in these cases is clear, is of assistance.

The symptoms of uremia may resemble those of meningitis; an examination of the urine, the presence of edema, the age and appearance of the patient, and the presence of albuminuric neuroretinitis would enable us to settle the question.

Care must be taken not to mistake the delirium which accompanies typhoid and other fevers for meningitis. Meningismus (p. 215) must also be considered.

Acute delirium or typhomania (p. 527) may be mistaken for meningitis. In this, optic neuritis, focal paralytic symp-

toms, muscular rigidity, hyperesthesia, and convulsions do not occur.

The distinctive features of serous, tuberculous, epidemic cerebrospinal and syphilitic meningitis will be mentioned when these forms are described.

The **prognosis** is very grave.

Treatment.—The patient must be kept quiet in a darkened room. A calomel purge should be given, followed by small doses of iodide of potassium and large doses of hexamethylenamin. An ice-bag should be applied to the head, and in robust adults blood may be abstracted by means of leeches or wet cups applied to the back of the neck or temples. The internal use of iodoform, gr. vj-xij daily, has been recommended. Favorable results have been reported from the use of inunctions of the shaved scalp with a 10 per cent. ointment of iodoform used conjointly with the above measures. Lumbar puncture may be of benefit. Pain must be controlled by bromides, phenacetin, or similar drugs, and if these fail by opium. In meningitis due to pus infection surgical measures (trephining) may be required. When due to influenza Flexner[1] has suggested the possibility of injecting a serum made from influenza bacilli, into the subdural space.

EPIDEMIC CEREBROSPINAL MENINGITIS OR SPOTTED FEVER

Definition and Etiology.—A specific infectious disease, occurring sporadically and in epidemics, caused by the Diplococcus intracellularis and characterized by inflammation of the cerebrospinal membranes and a clinical course of great irregularity (Osler).

Children are more liable than adults. It especially occurs in cold or temperate climates. It is most apt to attack those living in barracks or crowded houses.

Morbid Anatomy.—In malignant cases no change but extreme congestion in the membranes can be noticed, since

[1] Jour. Amer. Med. Assoc., July 1, 1911, p. 16.

death occurs before the characteristic lesions have time to develop. In ordinary forms the appearances are those of simple meningitis plus involvement of the spinal membranes. The Diplococcus intracellularis is found in the exudate, and secretions from the nose and throat.

Symptoms.—Malignant types occur during epidemics in which the patient is suddenly seized with chills, fever, headache, somnolence, and muscular spasms. The pulse soon becomes feeble and slow, and a purpuric rash appears, death occurring in a few hours.

In most cases the symptoms are similar to those of simple meningitis (p. 218), with the addition of pains in the back and limbs and the occurrence in most cases of a petechial rash. Urticarial, herpetiform, or erythematous eruptions may also appear. Leukocytosis also occurs. At the onset the pulse is full, strong, and slow. The elevation of temperature may be either slight or pronounced. Hydrocephalus may arise in the course of the disease due to the accumulation of fluid in the ventricles which in some cases at least is caused by blocking up of the foramen of Majendie. The following symptoms have been given by Koplik[1] as indications of the development of this condition: If after the initial stage has passed and the patient is apparently doing well, there is sudden dulling of the intellect, vomiting, dilatation of the pupils, strabismus, staring expression, and depression of the eyes showing the sclera. This may be further shown by percussion of the skull after the manner of MacEwen;[2] when the condition is present a tympanitic note will result. This, however, in children so young that the fontanelles have not yet closed is of no value, *i. e.*, under two and one-half years (Koplik) (p. 226). *Brudzinski's sign* is elicited by bending the neck, when flexor movements of the ankle, knee, and hip-joints occur. Passive flexion of one leg may also cause drawing up of the other leg. The pulse and temperature are irregular. Either pleurisy, pericarditis, parotitis, pneumonia, or arthritis may occur as complications.

[1] Amer. Jour. Med. Sci., April, 1907, p. 547.

[2] Pyogenic Diseases of the Brain and Spinal Cord, p. 148.

Diagnosis.—In sporadic cases the diagnosis may be difficult. Typhoid fever and pneumonia and other febrile diseases may present somewhat similar symptoms (p. 215). It may be still more difficult to distinguish it from other forms of meningitis. The most reliable test is the *lumbar puncture* (p. 221).

Prognosis.—The prognosis is grave, the mortality ranging in different epidemics from 20 to 75 per cent. The outlook is worse in children, and in cases having high fever, frequent convulsions, and deep coma. Cases of protracted duration do not often recover. Permanent palsies of cranial nerves, especially the auditory, may remain in the patients who do not die.

Treatment.—This consists of cold to the head and spine, blood-letting in the robust, cold packs or baths if the temperature is above 102°. Bromides, phenacetin, and similar sedatives should be given if opium is not used. As soon as the diagnosis is made, or before if the cerebrospinal fluid is cloudy, the antimeningitis serum of Flexner should be used. As much fluid is withdrawn as possible, and then inject 30 c.c. of serum if the quantity withdrawn is 30 c.c. or less. If more, inject, if possible, as much serum as fluid is withdrawn. This should be done daily until four doses are given. If the diplococci persist after this repeat the injections until they have disappeared. Hexamethylenamin should be given internally. The diet must be liquid and nutritious. Feeding by means of a tube introduced into the stomach may be necessary. Alcoholic stimulants must be given when the heart begins to fail.

POSTERIOR BASIC MENINGITIS

Etiology.—This is most frequently found in young infants. By many it is thought to be a sporadic and atypical form of cerebrospinal fever (p. 222).

Morbid Anatomy.—The meninges in the posterior fossa are thickened and adherent to the cerebellum and medulla. Hydrocephalus is frequently developed, as the exudation blocks up the foramen of Majendie (p. 230).

Symptoms.—These consist of vomiting, nuchal rigidity, opisthotonos, tetanic and epileptiform spasms, and frequently blindness due to pressure on the optic chiasm. The cranial nerves arising in the posterior fossa are frequently paralyzed.

Diagnosis.—The disease must be distinguished from tuberculous meningitis, which is often difficult. Lumbar puncture may aid (p. 227).

Prognosis.—Is grave, death frequently results, and if it does not the patient is usually left hydrocephalic.

Treatment.—The treatment advised for cerebrospinal meningitis (p. 224) should be employed. The internal hydrocephalus which may result has been treated by draining the fourth ventricle.[1]

TUBERCULOUS MENINGITIS

Etiology.—This affection, also known as *acute hydrocephalus*, is an infection of the meninges by the tubercle bacillus. It may occur at any age, but attacks children more often than adults, usually between the second and fifth years. A primary focus of tuberculous disease can usually be found, most often in the bronchial or mesenteric glands. The eruptive fevers, especially measles, and traumatism to the head sometimes seem to act as exciting causes. While the reverse is much more common, the process may extend from the spinal to the cerebral meninges.

Morbid Anatomy.—The arachnoid and pia at the base are mainly involved, the neighborhood of the Sylvian fissure, optic chiasm, and interpeduncular space being most markedly so. The membranes may be only slightly turbid and matted together; but usually there is a fibrinopurulent exudate covering the base and lateral surfaces of the brain. Tubercles appear as small whitish nodules on the membrane, especially about the Sylvian fissure. In some cases they can only be found upon the arteries at the base (the middle cerebrals

[1] Rev. Neurol. and Psychiat., January, 1911, p. 1.

and those of the perforated spaces). The cortex is usually also edematous and infiltrated with leukocytes. Areas of softening may occur in it. The ventricles are dilated and filled with turbid fluid. The spinal meninges are sometimes also involved. A few cases of tuberculous pachymeningitis have been reported.

Symptoms.—Prodromes are most common and of long duration (several weeks) in this form of meningitis. The patient loses flesh and is restless and irritable. The symptoms pointing to meningeal trouble may appear suddenly with convulsions; but more frequently the onset is insidious, the first symptoms being headache, which is intense, fever, and vomiting, which are followed by the other symptoms of meningitis (basilar form) already described (p. 220). In the paralytic stage the temperature may become subnormal. During the early stages the pulse is slow, becoming more rapid toward the close. Leukocytosis may also occur. Skene's sign, when present, is of diagnostic value. It is dependent upon a deposition of tubercles around the pupillary margin of the iris, which appears as a white wreath; in a few days a yellowish-brown circle takes its place, which gradually disappears as the dilatation of the pupil, common to the later stages, takes place. Choroidal tubercles may also be found in some cases. In adults the vertex is especially apt to be attacked and basal symptoms are frequently absent.

Diagnosis.—The diagnosis depends upon the discovery of a tuberculous focus elsewhere, or in the possession of a tuberculous heredity by the child, and the long duration of prodromes. Koplik[1] states that percussion of the skull, as recommended by MacEwen,[2] is of value in making an early diagnosis even before pronounced meningitic symptoms have appeared. In doing this the child should sit upright with the head slightly inclined to one side, the percussion being performed at the pterion. The greatly increased resonance caused by dilatation of the lateral ventricles will be found over the lower parietal bone.

[1] Jour. Amer. Med. Assoc., April 6, 1907, p. 1152. [2] Loc. cit.

A low temperature with meningitic symptoms points toward a tuberculous origin. The fluid withdrawn by lumbar puncture is clear, and often under high tension it may contain either tubercle bacilli or be apparently sterile. If the latter occurs when symptoms of meningitis are present, it is most likely tuberculous (pp. 96 and 97). A predominance of lymphocytes and the presence of Fehling's reducing substance is in favor of a tubercular cause (p. 98). In other forms of meningitis reduction does not take place. This may be the case also in tubercular meningitis if another infection is also present, as indicated by the presence of polynuclear cells, as these disappear the power of reduction returns[1] (p. 221). Meningitis due to infection from middle-ear disease or cerebrospinal meningitis may be mistaken for it (p. 221). The distinctive features between it and brain tumor are given on page 360.

The **prognosis** is most grave; recoveries, if they ever take place, are most rare.

Treatment.—This is similar to that of acute simple meningitis. Bleeding, of course, would not be indicated in this form. Small doses of potassium iodide frequently repeated have been extolled, as have also the inunction of 10 per cent. ointment of iodoform into the shaven scalp. As the subjects are ill-nourished, supporting treatment is indicated.

SEROUS MENINGITIS

Etiology and Symptoms.—Under this title Quincke has described a form of meningeal inflammation characterized by serous effusion into the membranes and ventricles. It is one of the causes of acquired internal hydrocephalus. The nature of the affection has not been made out. In some cases it is an inflammation of the ependyma. The onset may be sudden or gradual, and the course acute or chronic. It follows infectious diseases, alcoholism, chronic diarrhea, traumatism, and influences which depress or

[1] Kaplan and Casamajor, Archives Internal Medicine, February 15, 1912, p. 262.

deteriorate the general health. It occurs usually in childhood and youth. Fever is usually absent; if present it is slight and irregular. Headache is slight or sometimes absent, and rigidity of the cervical muscles is either absent or not severe. The symptoms usually present are mild delirium, insomnia, transitory attacks of stupor, vomiting, slow and irregular pulse, epileptiform convulsions, local palsies, especially of the cranial nerves, and optic neuritis, which occurs very frequently and often resembles the changes found in cerebral tumor (choked disk). Ataxia may also be a symptom. It must be remembered, however, that the symptoms may be as severe as those of other forms of meningitis. The alcoholic type frequently follows delirium tremens and is known as *post-delirious alcoholic stupor* or *alcoholic cerebral edema.* In these cases there is usually immobility of countenance, contracted pupils, some rigidity of the neck and other muscles, a muttering, inaudible delirium and hyperesthesia.

Diagnosis.—The acute form must be distinguished from purulent and tuberculous meningitis. The presence of fever, of a focus of suppuration elsewhere, would distinguish the former. The diagnostic features of the latter are mentioned on page 227. Lumbar puncture may be of service, as the fluid is clear in serous meningitis, cells few and bacteria absent, but the pressure is exceedingly high (p. 96). The preoccurrence of delirium tremens usually distinguishes the alcoholic type.

The *chronic* form may so resemble the symptoms of brain tumor, especially of the cerebellum, that it may often be impossible to distinguish the two. The symptoms following any of the causes mentioned would point to meningitis, while the presence of definite local symptoms, paralysis, or spasms would point to tumor.

Prognosis.—The prognosis is grave, but recovery may occur; even then hydrocephalus often remains. In the chronic form there may be remissions.

Treatment.—The treatment of the acute form consists of similar measures to those employed in other forms. Quincke advises mercurial treatment in all cases. The

chronic form may be treated by counterirritation to the back of the neck, mercurial or iodoform inunctions, and lumbar puncture. The alcoholic type should be treated with forced feeding and stimulation, strychnine and caffein being given in full and frequent doses.

CHRONIC CEREBRAL LEPTOMENINGITIS

Etiology.—This is usually secondary to acute attacks or brain tumor; but may be primary in syphilitics, alcoholics, after infectious diseases, in the insane, and following traumatism or sunstroke. A circumscribed serous meningitis also exists similar to that found in the spinal meninges (p. 217). It usually causes symptoms resembling cerebellar tumor.

Morbid Anatomy.—The membranes are thickened or often adherent to the cortex and dura. Exudations of lymph are present. The bloodvessels are thickened, and if the process is of long duration atrophy of the cortex is present. The appearances in syphilitic cases are described elsewhere (p. 408).

Symptoms.—The symptoms in general are those of acute meningitis, but are much less severe. There may be local symptoms, spasms, paralysis, etc., according to the situation of the inflamed area.

Diagnosis.—The disease may be confounded with brain tumor, chronic nephritis, hysteria, and neurasthenia. In tumor the symptoms are more intense and localized, optic neuritis, if present, is of a higher grade; but it should be remembered that the two may be associated. An examination of the urine and the presence of albuminuric retinitis will distinguish nephritis. The presence of cranial nerve paralyses, optic neuritis, and the previous history will enable one to distinguish between organic disease and hysteria and neurasthenia.

Prognosis.—The prognosis is unfavorable. It is best in those cases due to sunstroke, traumatism, or syphilis.

Treatment.—The treatment consists in the use of mercury and potassium iodide and persistent active counterirritation

at the back of the neck. Ergot and bromides are sometimes of service. Pain, insomnia, and impairment of the general health must be treated by the usual measures employed for such conditions.

HYDROCEPHALUS

This, while not always due to an inflammation, may be conveniently discussed here. Two forms are described, viz., external and internal. The former term is applied to an abnormally large collection of fluid outside the ventricular cavities (subarachnoid space). If alcoholic edema (p. 228) and serous meningitis are excluded, the condition is rare and is only found when an undeveloped brain fills in part only a cranial cavity of normal size. It may also rarely occur in connection with *internal hydrocephalus,* which is the common type and is that described.

Definition and Etiology.—It is a disease of infancy, characterized by gradual enlargement of the head and symptoms of brain irritation and of mental deficiency, and may be either congenital, also termed idiopathic, or acquired, also known as secondary; the etiology of the former is not well understood, but may be due to alcoholism, debility, or syphilis in the parents. It may occur as a familial disease, and it has been ascribed to fetal rachitis. The symptoms begin usually within the first six months of life; they may be present at birth. The acquired cases are due to anything which blocks up the passages between the ventricles and subarachnoid space (foramen of Majendie, foramina of Munro), as a basal meningitis of any type, especially the posterior form (p. 224), a tumor in the posterior fossa which makes pressure on the veins of Galen or aqueduct of Sylvius, cerebral tumors by pressure on the foramina of Munro, and inflammation of the ependyma and serous meningitis (p. 227). There is great dilatation of the ventricles and thinning of the cortex.

Symptoms.—The symptoms are a gradual increase in the size of the head (which becomes pear-shaped), but not of the face; bulging of the fontanelles; restlessness; irritability;

poor general nutrition; strabismus, and sometimes optic atrophy. The child either does not talk and walk or does so imperfectly. There may be spasticity of the legs. Finally, convulsions, which may be of the Jacksonian type, and coma occur, and death usually takes place in two or three years. If the secondary form develops after the sutures and fontanelles have closed a reseparation may occur, but in such cases the head does not become as large as when the condition develops in infancy. In tumors the occurrence of hydrocephalus may mask the localizing symptoms of the growth. Its existence may be determined by the finding of a "cracked-pot" percussion note when the skull is percussed.

Diagnosis.—The diagnosis must be made from rickets. In this the head is square, the fontanelles do not bulge, and other signs of the disease are present; and from brain tumor, which is often difficult, an *x*-ray picture may often aid, as the pressure of the cerebral convolutions may cause areas of atrophy of the bone.[1] (See also p. 360.)

Prognosis.—The prognosis is not good, but in mild cases of the idiopathic form the process may cease and adult life be reached.

Treatment is of not much avail. Mercury, potassium iodide, tonics, and nutrients may be given. Drainage of the ventricles has been done with good results in some cases.[2]

[1] Spiller, Rev. Neurol. and Psychiat., January, 1911, p. 8.
[2] Rev. Neurol. and Psychiat., January, 1911, p. 1.

CHAPTER X

SYSTEM DISEASES

CERTAIN diseases of the nervous system are confined either entirely or in greater part to one of the *tracts* or *systems of neurones* described on page 21 et seq. Thus, locomotor ataxia is a disease confined usually to the *sensory tract;* in some cases, however, motor neurones may also suffer. Progressive muscular atrophy is a disease of the *motor tract.* Such diseases are called *system diseases.*

DISEASES OF THE SENSORY OR AFFERENT TRACT

Posterior Poliomyelitis (Herpes, Herpes zoster, and Zona). —The ganglia upon the posterior spinal roots may become the seat of inflammation due to infection. Those upon the cranial nerves, as the geniculate, glossopharyngeal, auditory, and vagal ganglia, may similarly suffer, they being homologues of the spinal ganglia.

When the spinal ganglia are affected the **symptoms** are shooting, neuralgic pains in the course of the affected nerves, with sometimes areas of impaired sensation in the same area, followed by an herpetic eruption (p. 199). In the case of cranial nerves the similar symptoms occur in their course[1] (p. 165).

The **prognosis** in most cases is good, but disagreeable scarring may result and if the vesicles involve the eye blindness may follow.

The **treatment** consists in protecting the vesicles from rupture and infection by the use of anodyne dusting powders or salves, covering with a dressing. The salicylates and hexamethylenamin may be given internally.

[1] Hunt, Archives of Internal Medicine, June 15, 1910, p. 631

The constant current applied stabile in the course of the nerves may relieve the pain.

Locomotor Ataxia or Tabes Dorsalis.—**Definition.**—This disease, also known as posterior spinal sclerosis, is progressive, and is characterized usually by incoördination of movements, various sensory and trophic disturbances, and impairment of the special senses, especially the eye. It is due to disease of the posterior nerve roots, posterior columns of the cord, and at times of the spinal ganglia, of the peripheral nerves, and nerves of special sense.

The name "locomotor ataxia" is in general use, but, as will be seen below in the description of the symptoms, the disease may exist without noticeable ataxia being present. Hence, "tabes dorsalis" is a better name for the disease.

Etiology.—Locomotor ataxia is one of the most common of organic diseases of the nervous system. Males are more frequently attacked than females. A majority of the cases occur between the thirtieth and fortieth years, but may occur as early as ten or as late as sixty years of age. The most important cause is syphilis, about 75 per cent. of all cases having a history of this disease, the primary stage occurring ten to twenty years previously. It is a "parasyphilitic" disease, or one in which the patient has had syphilis, but in which the characteristic lesions of syphilis are not present and are not specially influenced by specific treatment. In other words, the probable way in which syphilis produces the disease is by its leaving a toxic principle of some sort in the blood which acts upon the sensory neurones, especially in the posterior roots, and causes them to degenerate; this degeneration not being different anatomically from that produced by other causes (p. 234).

Excessive fatigue, sexual excesses, infectious diseases, exposure to cold and wet are apparently causes in some cases.

Morbid Anatomy and Pathology.—*Macroscopically*, the following changes may be noticed: the pia mater between the posterior roots is thickened and opaque; the posterior roots may be either enlarged or in advanced cases thinner and more translucent; the spinal cord is reduced in size, and

the posterior columns appear shrunken and have a grayish appearance.

Microscopically, the first changes are found in the posterior roots, usually the lumbar, and the tract of Lissauer. Sclerosis will a little later be found in Burdach's columns, being first noticed along the median side of the posterior horns and spreading out toward the posteromedian septum as the disease advances upward, and, finally, involving the columns of Goll in the upper thoracic and cervical regions.

The fine fibers coming from the posterior roots, which run to the column of Clarke, are also sclerosed; but the cells themselves are usually not destroyed, and consequently the direct cerebellar tract is but rarely degenerated.

In *advanced cases* sclerosis of Gowers' tract may be observed. In rare instances the process may begin in either the sacral, upper thoracic, cervical, or bulbar nerve roots.

The bloodvessels of the cord show a small-celled infiltration of their sheaths and a diminution of their caliber.

The pathogenesis of the process is somewhat doubtful, but the most commonly accepted view is that the disease begins in the exogenous fibers (those that enter the cord from the posterior root ganglion). According to Nageotte, there is first an affection of the spinal roots between their entrance into the dura and the posterior ganglion (radicular nerve, Fig. 55). This consists of an endo- and perineuritis with interstitial and parenchymatous changes due to a mild but chronic syphilitic meningitis. The degeneration of the posterior columns is secondary and due to their fibers being cut off from their trophic centres (posterior root ganglia) by the pressure of the exudation. The nerve fibers as they pass through the pia have no neurilemma or sheath of Schwann, hence they cannot regenerate, Fig. 11. Orr and Rows[1] believe that the cause of the degeneration in this locality is not due to pressure, but to the influence of toxins brought through the lymph stream which flows in this situation.

[1] Brain, 1904, p. 460; Rev. Neurol. and Psychiat., May, 1907, p. 345; ibid., December, 1910, p. 721.

In addition to changes in the cord, others are more or less frequently found. The descending roots of the fifth and glossopharyngeal nerves may be degenerated. The optic nerve is frequently so affected. Other cranial nerves and their nuclei, especially the pneumogastric and motor nerves of the eye, may be also involved, respectively causing, when present, the visceral crises and palsies of the ocular muscles. The anterior horn cells and spinal nerves also may be found degenerated, which accounts for the muscular atrophy and weakness sometimes present. Valvular disease of the heart is frequently found.

FIG. 55

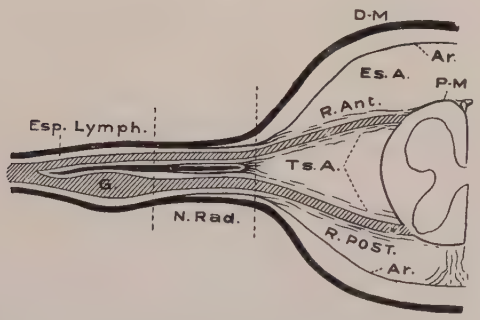


Diagram showing the relations of the meninges to the radicular nerve. *N.Rad.*, radicular nerve; *P.M.*, pia mater; *Ar.*, arachnoid; *D.M.*, dura mater; *R.Ant.*, anterior roots; *R.Post.*, posterior roots; *G.*, ganglion; *E.L.*, facultative lymphatic space. (From Nageotte.)

Symptoms.—These are best considered by dividing the progress of the disease into three stages, viz., the incipient or preataxic, ataxic, and paralytic.

The symptoms of the *incipient stage* are of special importance, as a recognition of the presence of the disease in this stage may be of the greatest value to the future welfare of the patient. One of the earliest is the finding of an increased number of lymphocytes in the cerebrospinal fluid and the Wassermann reaction. These may also be found in other lesions of the nervous system due to syphilis (pp. 97 and 241).

Pain is a frequent early symptom; it is usually characteristic, being sharp, shooting, and lancinating, and usually in the legs. In cervical tabes they are usually in the arms and in the bulbar form may be in the course of the trigeminal nerve. Dull pains resembling those of rheumatism may also occur. In some cases a girdle pain or sense of constriction about the body may be complained of. Various forms of paresthesia, as numbness and tingling of the feet, a sensation as if walking on feathers or something soft, are often present.

Visceral symptoms, or the so-called "crises," are rare—they may be present in this stage or may not occur until later. When present they consist of violent paroxysms of pain referred to various viscera, most commonly the stomach or larynx; but nephritic, rectal, urethral, and clitoral crises have been described. In the gastric crisis there are violent pains in the stomach, incessant vomiting, and the secretion of hyperacid gastric juice. The attack may last for days. It may consist of the pain alone. In the laryngeal crises there are dyspnea and noisy inspiration.

Often among the first symptoms complained of are difficulty in emptying the bladder or in starting the stream of urine; and decrease in sexual power and desire. Ocular symptoms, when they occur, are of great significance. They consist of (*a*) primary optic atrophy, evidenced by increasing loss of vision, sometimes ending in blindness; (*b*) paralysis of any or all of the external ocular muscles, evidenced by ptosis, single or double, or squint and diplopia—these paralyses are often transient, and a temporary diplopia may be the only symptom complained of; (*c*) a reflex iridoplegia or Argyll-Robertson pupil (pp. 65 and 145). The pupils are in most cases contracted (myosis), often unequally so. Alterations of the senses of smell and taste, either absence or perversion, may occur either as an early symptom or later.

One of the earliest and most significant symptoms is the *loss of the knee-jerk*. This occurs by a gradual decrease; it may be lost in one leg before the other, and often precedes for several years the development of the ataxia. In those

rare cases where the disease commences either above or below the lumbar enlargement it is preserved.

The Achilles-jerk is also usually lost early and in sacral tabes is lost when the knee-jerk may be present. In cervical tabes the tendon jerks of the arms, especially the biceps, are lost.

Trophic disturbances, as an arthropathy or perforating ulcer of the foot, are rarely early symptoms. These will be described later. The Romberg symptom and other evidences of ataxia (p. 55) may or may not be present in this stage; usually the application of the finer tests for ataxia (p. 55) will reveal its presence in some degree. Some cases never advance beyond this stage. This is especially true of those in which optic atrophy is an early symptom; such cases are known as the amaurotic or optic form (ocular tabes).

Sensory loss or diminution is frequently found early, especially in the chest, where it is apt to be a band in the nipple line, and in the legs and soles of the feet. One of the earliest symptoms is loss of the vibrating sense (p. 50).

Becoming physically easily exhausted and the presence of an increased abdominal reflex with absent knee-jerks have also been spoken of as preataxic symptoms.

Ataxic Stage.—One of the first symptoms of this stage is the inability to walk with facility in the dark. Examination will reveal the presence of the Romberg symptom, and the patient will respond imperfectly, or not at all, to the various tests. Gradually the characteristic gait appears: the patient walks with his eyes fixed upon the ground and the legs wide apart. The foot is raised high and thrown out with a sort of jerk, and is brought down hard, with the heel usually first. In the course of time assistance in the shape of one or two canes has to be employed (Fig. 56). Incoördination in the movements of the hands and arms may or may not be present; in those cases in which the cord is first affected high up they are involved first.

In this stage there is no loss of muscular power, but there may be great muscular relaxation, which permits greater mobility of the joints than normal (Fig. 57).

The sensory symptoms that occur at this time are varied.

The shooting pains may persist or may be absent. Bands of anesthesia about the body, following the sensory distribution of the spinal segments, may be found (segmental or

FIG. 56

FIG. 57

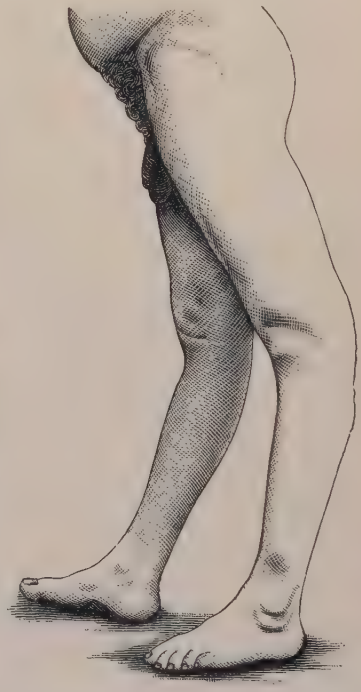


FIG. 56.—Gait in tabes. Observe the overextension of the supporting knee, rigidity of advancing leg, elevated toe, heavily descending heel, watchfulness of steps, and assistance by cane. (Church and Peterson.)

FIG. 57.—Abnormal position of the knees in standing in locomotor ataxia. (Dejerine.)

spinal type, see Spinal Localization). In the legs appreciation of painful stimuli is usually delayed, and sometimes there is inability correctly to localize sensory impressions (false allochiria, p. 49).

Muscle sense, especially the inability to recognize position,

becomes much impaired. Paresthesiæ, such as have been previously described, persist.

The knee-jerks remain as in the preataxic stage, while the skin reflexes are often increased.

Fig. 58 Fig. 59

Fig. 58.—Disease of both knee-joints and both ankles in locomotor ataxia.
Fig. 59.—Perforating ulcer of the foot in locomotor ataxia. (Obersteiner.)

The Argyll-Robertson pupil persists, and optic atrophy is usually found; but as has already been stated, cases in which this symptom is marked frequently do not reach this stage. Deafness, which may be associated with attacks of vertigo, due to degeneration of the auditory nerve, rarely may develop.

Crises are more apt to occur in this period than in the preataxic. Constipation is common. Retention of urine,

with the development of cystitis, may appear and the sexual power grows weaker.

It is in this period of the disease that *trophic symptoms* are most apt to occur: herpes sometimes appears in the course of the shooting pains (p. 232). The nails become thickened, ridged, and brittle, and onychia may develop. A perforating ulcer, usually located beneath the big toe, may appear (Fig. 59). Lesions of the joints, the so-called arthropathies, or Charcot's joints, when present usually affect the knee, but other joints may suffer. They consist of painless swellings, due to effusion into the joint, attended with destruction of the cartilages and bones, and possibly suppuration, causing dislocation and deformity (Fig. 58). The bones may become brittle and liable to fracture from the slightest causes. Muscular atrophy, sometimes of the Aran-Duchenne type (p. 269), and due to degeneration of the cells in the ventral horns,[1] sometimes occurs. Atrophy of muscles may also be due to degeneration of the motor nerve roots (p. 235).

In this stage mental symptoms sometimes develop; paretic dementia sometimes coexists, either following the appearance of the tabes or *vice versa*. Either melancholia or simple dementia may also develop in the course of the disease.

After the symptoms above described have persisted for an indefinite period, from three to thirty years, and the patient does not die of some intercurrent malady, the symptoms of the *paralytic stage* usually develop, in which muscular weakness makes its appearance, and the patient gradually loses the power of walking and becomes bedridden. In this condition the patient may live a long time.

Diagnosis.—The early recognition of the trouble is of great importance. Loss of either knee-jerk, Achilles-jerk, or biceps-jerk, associated with one or all of the following symptoms—viz., lightning pains, sensory loss or diminution as described on page 237, optic atrophy, ocular palsies, the Argyll-Robertson pupil, loss of sexual power, bladder weak-

[1] Lapinsky, Archiv f. Psychiatrie und Nerven Krankheiten, vol. xl, part 3. Wilson, Rev. Neurol. and Psychiat., August, 1911, p. 401.

ness—should justify a provisional diagnosis, which is rendered still more positive if a history of syphilis can be obtained. All of the symptoms above described do not occur in every case of ataxia. Many of them rarely do. An examination of the cerebrospinal fluid is of importance in doubtful cases. An increased number of lymphocytes will be found, the Fehling reducing substance will be present, and the globulin is often not increased (p. 97). The Wassermann reaction may be absent, especially if the patient has had antisyphilitic treatment, but is present in the serum of most cases. According to the experience of Kaplan and Casamajor,[1] it is not so often found in the cerebrospinal fluid. It is more likely to be found in both serum and fluid in paresis (p. 417), and to a less extent in cerebrospinal syphilis. Thus in 60 cases of paresis studied by these authors the reaction was positive in both serum and fluid in 100 per cent. In 46 cases of cerebrospinal syphilis it was present in both in 15 cases and in the serum alone in 28. In one it was only found in the fluid and 2 were negative in both. In 60 cases of tabes it was present in both in 6 cases. In 32 it was only present in the serum and in 8 it was only present in the fluid, 14 were negative in both. It should be stated that other observers have obtained somewhat different results.

A syphilitic leptomeningitis involving the posterior nerve roots may cause symptoms that clinically cannot be distinguished from true tabes. Examination of the cerebrospinal fluid (p. 412) may, however, often enable the distinction to be made. Mott[2] states that in true tabes the lymphocytosis does not diminish with antisyphilitic treatment, and in syphilitic pseudotabes it does. The percentage of cells in the fluid is much greater in cerebrospinal syphilis (p. 418), and the globulin is always + (Nonne's Phase I).

When the disease is fully developed it may be confounded with the following diseases:

Multiple neuritis, which differs from tabes in the early development of motor paralysis and muscular atrophy,

[1] Archives of Internal Medicine, February 15, 1912, p. 262.

[2] Abstract in Rev. Neurol. and Psychiat., May, 1910, p. 315.

tenderness of the nerve trunks, and constant pain, aggravated by movement. *Diphtheritic neuritis*, in which sensory symptoms are usually absent or slight; but the presence of paralysis of the muscles of the throat and the history should make the diagnosis clear. *Diabetes* may cause symptoms which closely simulate tabes. Loss of knee-jerk and Achilles-jerk, impotence, pains in the legs and perforating ulcers may be so caused. The pain is usually not shooting in character as in tabes, the urine will contain sugar, the calf muscles are usually hyperesthetic, while girdle pain and zones of diminished sensation, Argyll-Robertson pupil, arthropathies, and crises do not occur in diabetes.

Ataxic paraplegia (p. 287), which differs in the increase of knee-jerks, spasticity of the muscles, absence of pain and eye symptoms.

Diseases of the sensorimotor region of the cerebral cortex, especially in the superior parietal lobule and of the sensory tracts, may cause ataxia, which, however, is usually limited to one limb, and is accompanied with other symptoms of cerebral disease. Also in spinal ataxia the movements become better regulated when under the control of the eyes; in cortical ataxia they do not.

Cerebellar disease, if a growth, presents the general symptoms of brain tumor—nausea, optic neuritis, headache. No matter what the cause the gait differs, being reeling, with a tendency to fall in one certain direction, resembling that of a drunken man, instead of the gait described as ataxic (pp. 55 and 237). There are usually no sensory symptoms.

General paresis may be associated with tabes, either preceding it or coming on in its later stages. The peculiar mental and physical symptoms which are symptoms of paresis will enable one to recognize its presence (p. 415).

Visceral crises, especially when they occur as early symptoms, may be mistaken for disease of the affected part. Paroxysmal attacks of visceral pain should cause an examination to be made for other symptoms of tabes.

Prognosis.—As has been already stated, locomotor ataxia usually lasts a long time. Rarely it may run its course rapidly, the paralytic stage being reached in a year or less.

Many cases remain in the preataxic stage for years. Some, especially those associated with optic atrophy, never advance beyond it. Those cases in which there is a marked lymphocytosis and Wassermann reaction in the serum but not in the cerebrospinal fluid are most amenable to treatment (Kaplan and Casamajor).

While complete recovery rarely occurs, much can be done to ameliorate the symptoms and to lengthen the period before the paralytic symptoms occur.

Treatment.—It is well that all cases in which the cerebrospinal fluid shows a marked lymphocytosis (64 to 98 per c.mm.) and the Wassermann is positive in the serum should receive a vigorous antisyphilitic treatment (salvarsan and mercury). Cases in which the Wassermann reaction is negative are not suitable for antisyphilitic treatment (Kaplan and Casamajor), and even in those considered suitable good results are often not obtained. The following measures may then be tried; long-continued courses of nitrate of silver (gr. j, t. i. d.), arsenic, chloride of gold and sodium, and glycerophosphates of lime and soda. Small doses of strychnine in cases associated with weakness are often beneficial. Where there is much difficulty in urinating it is of special service. In addition to the use of drugs, the patient must be cautioned not to overexert himself physically or mentally; to abstain from alcohol, excessive use of tobacco, and sexual indulgence; and he should have plenty of nutritious, easily digested food, fresh air, and sunshine. A warm, dry climate is preferable. In the early stages or in greatly debilitated cases the rest cure (p. 93) is often of great benefit. Hydrotherapeutic measures may be tried in the early stages. A most valuable plan is to cause the patient to sit from twenty to thirty minutes in water of a temperature of 90°.

The back during this time may also be douched for a few minutes with water alternating from 75° to 95°. After the bath the patient should be well rubbed with warm towels.

Massage, independently of the rest cure, may be of value early in the disease and when hypotonia is marked.

Of the various symptoms which need relief, *pain* is one of the most important. The various analgesic coal-tar

preparations, either alone or in combination with codeine, often answer. Salvarsan in some cases causes improvement. Rest in bed and counterirritation by blister or cautery over the spine may be tried. When these fail suspension of the body from the shoulders and head has often proved valuable for this purpose. A similar effect may be obtained without apparatus by placing the patient in a sitting position with the legs held straight down in the extended position, then gradually flexing the body upon the legs and keeping it so for several minutes. Suspension must not be employed where heart or arterial disease is present, or where there is debility, and should only be employed for a short interval (thirty seconds) at first, which period may be gradually increased to five minutes or longer. To relieve the numbness and tingling of the legs, electricity, in the form either of faradism applied by the dry brush, or the static breeze or high-frequency current, is useful. Bandaging the legs has also given relief. In laryngeal crises a few whiffs of chloroform or the inhalation of nitrite of amyl will usually suffice. In the other forms of crises the coal-tar preparations already mentioned are indicated. In the gastric crisis cocaine, carbolic acid, oxalate of cerium, heroin, and codeine should be tried. As a last resort only should morphine be used. In extreme cases the seventh, eighth, and ninth posterior spinal roots may be cut. Osler recommends, in all cases associated with increased arterial tension, the long-continued use of nitroglycerin for the relief of pains and crises. The bladder is liable to become infected and should be carefully watched; catheterization and irrigation at regular intervals combined with the internal use of hexamethylenamin should be practised if any symptoms of disturbance are present.[1]

Frenkel's method of exercise and muscle reëducation described on page 88 should be employed when the ataxia is pronounced. Frequently, also, support of the arch of the foot, as for flat-foot, may be required.[2]

[1] Barney, Boston Med. and Surg. Jour., December 22 and 29, 1910, January 5, 1911, pp. 993, 985, and 13.

[2] Schwab, Jour. Amer. Med. Assoc., December 16, 1905, p. 1840

DISEASES OF THE MOTOR NEURONES

Diseases of the motor neurones may be limited to either the *central* or *peripheral* neurones, or both may be attacked.

DISEASES OF THE CENTRAL MOTOR NEURONES

Primary Lateral Sclerosis.—Definition.—A spastic paralysis of the muscles of the body, those of the legs being first and most affected, attended with increased reflexes in the affected parts and unattended by muscular atrophy or sensory disturbances.

Etiology.—Primary lateral sclerosis occurs most commonly between the ages of twenty and forty, but may appear earlier or later. It is a rare condition and by some believed not to exist, the cases so called being either a chronic myelitis (p. 387) or atypical form of multiple sclerosis (p. 404). The causes are not well understood. Some cases have seemed to follow syphilitic infection (Gowers); others have appeared to be due to traumatism to the back, exposure to cold and wet, infectious diseases, and to causes producing general physical debility. There is an *hereditary spastic spinal paralysis* which presents the symptoms of lateral sclerosis. I have seen three cases: the father and two sons. In this family there have been fourteen others, extending through different generations, who have had similar symptoms. These cases may develop quite early (eighteen months in one of the above).[1] (See p. 255.)

Morbid Anatomy.—The lesion is a degeneration of the pyramidal tracts. Many observers doubt if a degeneration of the pyramidal tracts can occur excepting as a *secondary* degeneration due to a lesion higher up, cutting off the fibers from their parent cells in the cortex; or to a lesion of the cells themselves. A few cases have, however, been reported in which no such lesion could be found.

Symptoms.—The patient first complains of soon tiring and a feeling of stiffness in the legs, and there may be dull pains

[1] Spiller, Philadelphia Med. Jour., June 21, 1902.

in the back. The symptoms may commence in one leg before the other is affected, or one leg may be affected more than the other. The loss of power in most cases increases very gradually, and at the end of years weakness may not be extreme. The spasticity of the muscles increases more rapidly; the affected limbs, when bent by the examiner, may at first give a sensation similar to that given by the bending of a piece of lead pipe; but if the movement is continued, they become more supple. In the early stages the only thing noticeable about the gait is a tendency to drag the toes and a rapid wearing off of the toes of the shoes. The gait in a well-marked case is characteristic; the legs are moved stiffly and seem glued to the ground, being pushed forward; the toes are raised over obstacles either with great difficulty or not at all. As the patient begins to walk a violent trembling or clonus of the legs often occurs. Owing to spasm of the adductors, the knees are kept close together; and in aggravated cases it may cause the patient to walk with one leg crossed over the other, the so-called cross-legged progression (Fig. 61). The knee-jerk is markedly increased and the ankle clonus and Babinski reflex well developed. The superficial reflexes are also more active. The muscles are not atrophied and feel firm; electrical reactions are normal. There may be difficulty in holding the urine, but usually there is no trouble with the sphincters until late in the disease. Sensory disturbances are absent. As the disease progresses the arms may also become affected. In the hereditary types pes cavus and talipes varus may develop.

Diagnosis.—The following diseases resemble primary lateral sclerosis:

Transverse myelitis; in this, however, there are usually sensory symptoms, as pain and anesthesia, and the sphincters are early affected.

Ataxia paraplegia differs in the spasticity of the muscles not being so marked, and evidences of lack of coördination are present; involvement of the sphincters is also common.

In *amyotrophic lateral sclerosis* there are soon developing muscular atrophy and weakness.

Secondary degeneration following *cerebral apoplexy* may be mistaken for the cases in which one side is more affected than the other. The history of a previous apoplectic attack with sudden onset of the paralysis, which at first may be flaccid, will settle the question.

Hysterical paraplegia may be often most difficult to distinguish. In lateral sclerosis the spasm is most pronounced when the limbs are extended; as they are flexed it diminishes, a condition which does not occur in hysteria. Also, the deep reflexes, while they may be increased, are not so much so in hysteria, a true ankle clonus being exceedingly rare in this disease. The Babinski reflex is present in lateral sclerosis but is not in hysteria. Other evidences of hysteria will also be found if searched for.

The **progress** of the disease is steadily but slowly onward, until the power of movement is entirely lost.

Treatment.—This is not very satisfactory. Nitrate of silver, arsenic, chloride of gold and sodium, iodide of potassium, and bichloride of mercury may all be tried. Rest may prove beneficial, and prolonged warm baths relieve the muscular rigidity. Massage may also prove of service. Strychnine must not be given. If motor paralysis is not extreme but the chief cause of disability is spasticity, cutting some of the posterior spinal roots may be considered. This has given good results in some cases.[1]

In the hereditary cases I have seen relief after proper tenotomies to relieve the deformity, and the use of faradism to the weaker muscles (peroneal and extensor group).

Unilateral Ascending and Unilateral Descending Paralysis.—In 1899 Mills[2] called attention to a symptom group presenting the symptoms of a spastic paralysis (disease of central neurone), but in which the symptoms began in the lower extremity and progressively ascended and also were confined to one side. Since this a number of such cases have been reported. Mills, in a review of these cases,[3] says that this type may be produced. (1) By primary degeneration of

[1] New York Med. Jour., January 29, 1910, p. 215; Jour. Amer. Med. Assoc., January 15, 1910, p. 210.

[2] Jour. Nerv. and Mental Dis., April, 1900.

[3] Proceedings Neurological Section of Amer. Med. Assoc., 1906, p. 166.

the pyramidal tracts to which may be added other degenerative lesions; (2) as the early stage of multiple sclerosis; (3) as the form assumed by unilateral amyotrophic sclerosis; (4) as the order of progression in unilateral paralysis agitans; (5) as the expression of a focal lesion either cerebral or spinal; (6) as a clinical type in cerebrospinal syphilis; and (7) as a peripheral or hysterical affection.

Similar causes have also caused a progressive unilateral descending paralysis.

Diagnosis.—The diagnosis of the cause of these symptoms can be made by paying attention to accompanying symptoms. Thus if *multiple sclerosis*, some of the classical symptoms of that disorder will likely be present; if amyotrophic lateral sclerosis, atrophy of muscles and fibrillary tremors will be found; *paralysis agitans* will have the characteristic facial expression; the Babinski reflex will not be present; tremor may or may not be a symptom (p. 422). A *tumor* might begin in the leg centre and then progressively involve other centres, but some of the general symptoms of tumor would be present and Jacksonian epileptic seizures would sooner or later appear. Necrotic areas, due to *arteriosclerosis*, might also cause such symptoms, but the progression would not likely be so regular, and other symptoms of vascular disease could be found. *Syphilis* may cause such a symptom group, but the history and existence of other evidences of the disease will differentiate it. If due to a peripheral lesion other signs of such a lesion, as absent reflexes, changes in electrical reactions, pain, etc., will be present. If due to *hysteria* other stigmata of that disease would be found; the Babinski reflex would be absent. If these various conditions can be excluded the symptoms are probably due to a slowly increasing degeneration of the pyramidal tracts or of the cerebral motor neurone system.

Progressive Lenticular Degeneration.—Under this title Wilson[1] has described a rare disease, which may attack more than one member of a family, but is not hereditary. It always occurs in young people and may be either acute or chronic. The symptoms are bilateral tremor

[1] Brain, 1912, p. 295.

of all the limbs and sometimes the head which is increased by volitional movements; spasticity of the limbs and face; dysphagia and dysarthria and finally anarthria; sometimes spasmodic laughter and emotionalism; some difficulty in maintaining the equilibrium due to the spasticity; sometimes transitory mental symptoms. The reflexes are normal and there is no motor weakness, Cirrhosis of the liver is always associated, but no signs of liver disease are present during life. A bilateral symmetrical softening of the lenticular nucleus, involving especially the putamen, is found. The internal capsule is intact. He believes it to be a pure syndrome of the corpus striatum (p. 302). The disease is not thrombotic, the change, however, begins around the lenticulostriate vessels, as a result of which the cells and fibers of the lenticular nucleus gradually disappear and their place is taken by glial tissue. In advanced cases the nucleus may break down and cavity formation result. Wilson believes it to be due to a toxin which probably has some relation to the cirrhosis of the liver. The disease is fatal in from six months to five years.[1]

SPASTIC PARALYSIS OF INFANTS OR CEREBRAL PALSIES IN CHILDREN

This may involve all four extremities (*diplegia*); or be *paraplegic* or *hemiplegic* in its distribution. The cases due to apoplexy (p. 253) are not true system diseases, as other parts of the brain than the motor tracts may be involved. For convenience, all forms will be here considered.

Spastic Hemiplegia.—Etiology.—This form usually sets in during the first or second year; cases occurring after five years are rare. Neurotic taints, alcoholism or syphilis in the parents in rare instances appear to act as predisposing causes. A small number of cases, those occurring soon after birth, are due to difficult labor and injuries from the forceps. The most common cause is one of the infectious fevers. Traumatism to the head is a rare cause.

[1] Dana (Jour. Nerv. and Ment. Dis., February, 1908, p. 65) has reported bilateral softening of the lenticular nuclei due to gas poisoning. McConnell and Spiller (Jour. Amer. Med. Assoc., December 14, 1912, p. 2122) have also reported a case in which a similar condition was found.

Symptoms.—In those cases due to injury at birth the paralysis may be noticed immediately or soon after birth. This cause, however, more commonly produces diplegia or paraplegia (p. 251). The onset in most cases of hemiplegia is sudden and attended with loss of consciousness and partial or general convulsions lasting from an hour or so to days. Fever may be present. More rarely paralysis suddenly occurs without loss of consciousness or convulsions. When the child recovers consciousness, paralysis more or less complete of one side is noticed. The face usually escapes.

Fig. 60

Spastic hemiplegia with epilepsy (Philadelphia Hospital).

In a few cases this paralysis grows less as the child advances in years, and in course of time may almost disappear. In the majority of cases a well-marked hemiplegia is left. Motion is more or less impaired and the muscles become spastic, the arm is carried with the forearm flexed upon the arm and the fingers contracted (Fig. 60). The gait is that characteristic of hemiplegia, the toes being dragged and the leg swinging from the hip in the arc of a circle. The reflexes are increased. If the lesion is upon the left side of the brain, aphasia may be a symptom. This is rarely permanent. Occasionally in time the muscles lose their rigidity or become more relaxed than normal (hypotonia), and the reflexes may be normal or diminished. There is usually arrest of development or retardation of growth in the limbs, which are consequently smaller than those of the other side. Vasomotor disturbances, as coldness, blueness, and congestion of the affected parts, occur. Arrest of mental development occurs in a large number of these patients, and all the various grades of idiocy and imbecility may be found among them.

Epilepsy is also a frequent sequel, 41 out of 135 in Osler's series. The seizures may be either of the general, Jacksonian, or petit-mal type.

The so-called posthemiplegic movements occur more or less frequently. These are tremor, ataxia of the affected limbs, sometimes spoken of as posthemiplegic chorea, and athetosis, which has been described on page 37.

Spastic Paraplegia and Diplegia.—Etiology.—This affection is also known as *Little's disease* and *birth palsy*. The majority of cases of hemiplegia are not congenital, but have their onset a year or two after birth. In the form about to be described the opposite is the case, only a limited number occurring any length of time after birth, and the majority being congenital in origin. The majority of the children so affected are born in first and difficult labors, or have been forceps deliveries. A certain number of others have been premature births, and the symptoms are due to lack of development of the pyramidal tracts. Neurotic taints, alcoholism, and syphilis in the parents may in these latter cases rarely have some influence. Injuries to the abdomen or the occurrence of infections or exhausting disease during pregnancy of the mother have also appeared to be causes in some cases.

Symptoms.—In view of the fact that Cushing[1] has advised operation in the acute stage of this condition and has done so in four cases with favorable results in two, it is important that the earliest symptoms be recognized. These are tense and bulging fontanelles, slowing of the pulse, asphyxia, inequality of the pupils, and blood corpuscles in the fluid obtained by lumbar puncture. In some instances nothing abnormal is noticed about the child until it is time for him to begin to walk, which he either does not attempt at all or else does so with difficulty. Often the child will be unable to sit up, and the head rolls about owing to its not being supported by the neck muscles. Inquiry will often elicit the fact that there have been a number of convulsions shortly after birth. In well-developed cases the legs are rigid, and

[1] Amer. Jour. Med. Sci., October, 1905, p. 563.

when stood upon its feet the weight is supported upon the toes and inner surface of the feet; the knees are held close together, owing to adductor spasm (scissors gait), or the legs may be crossed (Fig. 66).

The rigidity of the *arms* when they are affected varies. It may be slight, and is evidenced principally by awkwardness in using the hands; or it may be considerable, and the arms are held in the position assumed by the arm of a hemiplegic. The face is but rarely affected.

Fig. 61

Spastic paraplegia; cross-legged progression (Jefferson Hospital).

The legs are usually more affected than the arms. The cases in which the arms are so slightly affected as to be only appreciable after careful examination are known as the paraplegias. Spasticity is sometimes absent. The reflexes are increased and the muscles not wasted; sensory disturbances are absent. The head is often microcephalic. Irregular muscular movements are often present. These consist either of constant movements of the muscles which interfere with attempts at grasping an object and similar movements of the shoulders, arms, and sometimes the head; or of athetoid movements (p. 37), which in these cases are most pronounced. These are bilateral, involving both hands and feet, and are often associated with irregular movements of the facial muscles, causing grimaces and distortion of the features.

Epileptic convulsions occur in many of the cases. Intelligence may be good, but many of these patients are idiotic.

Cerebral Palsies of Children.—Morbid Anatomy.—In an analysis by Osler of ninety autopsies in cases of hemiplegia there was found either hemorrhage, thrombosis, or embolism of a Sylvian artery in sixteen cases.

The diplegias and paraplegias which make their appearance shortly after birth, and also the rare cases of hemiplegia which occur at this time, are due to meningeal hemorrhage either from a vein or artery, usually the former. The hemorrhage usually being over the motor region of one or both sides. Some cases may be due to fetal meningoencephalitis.

The cases which occur in children born prematurely are often due to non-development of the pyramidal tracts. Some cases may be due to "agenesis corticalis," or a defective development of the cellular elements of the entire cortex.

If the patient lives for a number of years the following lesions may be found; most frequent is atrophy and sclerosis which may involve either a group of convolutions, an entire lobe or the hemisphere. The affected parts are firm and hard and the convolutions smaller than normal. The sclerosis may be diffuse (p. 406) and there may be nodular projections (hypertrophic sclerosis).

Porencephalus is found next most frequently. By this is meant a loss of brain substance which causes either a cavity or cyst which may communicate with the ventricle. Porencephalus may be due either to hemorrhage occurring at birth, lack of development or the lesions of apoplexy (p. 322) which may occur after birth. In atrophy and sclerosis the primary lesion is doubtful, Strümpell believes they are due to cortical polioencephalitis (p. 336).

The *following table* from Sachs shows the lesions most likely to be present, according to the time of onset:

Groups.		Morbid lesion.
Diplegia and paraplegia.	Paralysis of intra-uterine onset.	Large cerebral defects; porencephaly. Defective development of pyramidal tracts. Agenesis corticalis (highest nerve elements involved).
	Birth palsies.	Meningeal hemorrhage, rarely intracerebral hemorrhage. Later conditions, meningo-encephalitis chronica, sclerosis, cysts, partial atrophies.
Hemiplegia.	Acute (acquired) palsies.	Hemorrhage, meningeal and rarely intracerebral; thrombosis, from syphilitic endarteritis and in marantic conditions; embolism. Later conditions, atrophy, cysts, and sclerosis (diffuse and lobar). Meningitis chronica. Hydrocephalus (seldom the sole cause), primary encephalitis, polioencephalitis acuta (Strümpell).

Diagnosis.—These affections are sometimes mistaken for acute poliomyelitis, but a knowledge of the differences in the character of the paralysis due to lesion of the central and peripheral motor neurones will at once distinguish the two (p. 42). There may be more difficulty in cases of mild type, in which rigidity and contractures are but slight or absent and the reflexes not increased; but the absence of complete or partial De R, the distribution of the paralysis and mode of onset, will distinguish the cerebral from the spinal paralysis.

Patients in whom choreiform or athetoid movements are present, and in whom the evidences of paralysis have pretty well disappeared, may be mistaken for chorea. Attention to the history, the presence of increased reflexes, evidence of slight paralysis or rigidity, the peculiarity and rhythm of the movements in athetosis, will indicate the nature of these movements. Also the rigidity sometimes present in rachitic children may be mistaken for the diplegic form. In the former the spasm often begins in the hands, and is confined to the hands and arms. The spasms may be painful, and are intermittent; the laryngeal muscles are often affected, producing difficult respiration (laryngismus stridulus). (See Tetany.)

Prognosis.—The prognosis, as regards recovery of mental and physical strength, is bad. Many of the cases which survive the attack either develop epilepsy, become idiotic,

or are helpless cripples. Some do not, but the possibility of this latter result occurring cannot be told until the child has been under observation for some time. The non-appearance or diminution in frequency of convulsions and the absence of contractures are favorable signs. Aphasia, if present, usually disappears.

Treatment.—In the acute stages the physician will usually be called on account of the convulsions. In these cases he should endeavor to control them with chloroform and afterward administer bromides; move the bowels with calomel, enjoin rest and quiet, and apply cold to the head. If any of the symptoms detailed on page 251 are present, and the labor has either been prolonged or instrumental, the question of opening the skull, as advised by Cushing,[1] should be considered. One of his patients, in which favorable results were obtained, was not operated upon until eight days old.

The treatment of the resulting paralysis consists of the use of electricity to the extensor muscles and massage of the same after the acute symptoms have disappeared (about two weeks after onset); and overcoming the tendency to contractures by passive movements. If these have already developed, the orthopedic physician may do much by proper tenotomies and the application of suitable braces. When paralysis is not extreme but spasticity is, or athetoid and other spasmodic movements are severe, cutting some of the posterior roots (p. 247) may be of service.[2] The epilepsy is treated similarly to idiopathic epilepsy (p. 442). By special methods of education, as practised in institutions for feeble-minded children, the mental condition may often be improved.

Herditary Spastic Paralysis.—Definition.—This is a rare condition affecting a number of members of a family in the same and different generations. Some of the cases at least appear to be due to hereditary syphilis. The cases may assume a spinal or cerebral type.

[1] Loc. cit.

[2] Clark and Taylor, New York Med. Jour., April 13, 1912, p. 729.

Morbid Anatomy.—The spinal type appears to depend upon a degeneration of the spinal portion of the pyramidal tract (lateral columns).

The principal changes found in the *cerebral* type have been small, poorly developed cerebral convolutions, degenerative changes in the cells of the cortex and medulla, corpora quadrigemina, geniculate bodies, third and fourth cranial nuclei, and in the cells and fibers of the optic tract. Changes have also been found in the pyramidal tracts and cells of the gray matter of the cord. It has been thought by some that these changes are due to a toxemia.

Symptoms.—*Spinal type;* in brief, the development in infancy or childhood of spastic rigidity and paralysis with increased reflexes, affecting principally the legs and without cerebral symptoms—*i. e.*, mental deterioration, epilepsy, etc. (p. 251).

A group of cases differing somewhat from these has been described by Bernhardt and Strümpell, in which the symptoms do not begin until between twenty and thirty. At first there is no paralysis, but rigidity of the legs. After a long period the arms may be also affected. Toward the end paralysis of motion and sensation may develop.

Cerebral Type.—These cases are usually known as *amaurotic family idiocy* or Tay-Sachs disease. The degeneration is not confined strictly to motor neurones, but it can be more conveniently described here. The symptoms, as summarized by Sachs, are: psychic disturbances that appear in early life (six months or a year) and progress to idiocy; weakness and ultimately paralysis of the extremities, which may be either flaccid or spastic; increased, decreased, or normal reflexes; partial followed by total blindness due to peculiar changes in the macula and atrophy of the optic nerve; marasmus and death by the end of the second year. It is a family disease. Occasionally nystagmus, strabismus, and deafness may be present. Most of the reported cases have been in Jews.

Diagnosis.—The diagnosis must be made from cerebral spastic paralysis due to hemorrhage. From this the *spinal* form differs in its gradual development, the occurrence of

similar symptoms in other members of the family, absence of initial convulsions, and cerebral symptoms. The *cerebral* type will be distinguished by its gradual onset, absence of convulsions, the development of blindness. Somewhat similar symptoms to the cerebral form may be caused by hereditary syphilis.

Prognosis.—The prognosis is sufficiently indicated in the account of the symptoms.

Treatment. — The treatment is evidently symptomatic (p. 255). Hirsch, taking the ground that the cerebral form is due to a toxemia, recommends removing the child from the breast and preventing the mother from so feeding any future children.

DISEASES OF THE PERIPHERAL MOTOR NEURONES

Acute Anterior Poliomyelitis.—Definition.—This disease, also known as infantile paralysis, is of infectious origin and usually occurs during early childhood; it is characterized by fever, general loss of power, followed by recovery of most of the affected muscles, while those in which paralysis remains rapidly atrophy.

Etiology.—The disease is most common in the warm months. The nature of the infection so far is unknown.[1] The disease has been produced in monkeys by intracerebral inoculation with an emulsion of an affected spinal cord.[2] Monkeys have also been inoculated with the nasopharyngeal secretions of patients suffering from the disease, and Flexner, Clark, and Dochez[3] have demonstrated that the virus will live in the stomach and intestines and pass from the body in the feces. Infectious diseases, cold, traumatism, overexertion, and dentition may be predisposing causes. Extensive epidemics of the disease have occurred. While more common in young children, adults may be attacked.

[1] Recent investigations show that the stable fly (Stomoxys calcitrans) plays a part in the transmission of the disease. (Jour. Amer. Med. Assoc., Nov. 2, 1912. p. 1627).

[2] Flexner and Lewis, Jour. Amer. Med. Assoc., January 1, 1910, p. 45.

[3] Jour. Amer. Med. Assoc., July 27, 1912, p. 273.

Symptoms.—In the majority of instances the patient, having previously been in good health, becomes feverish, restless, and may complain of headache and anorexia. There may be general cutaneous hyperesthesia. Convulsions also may rarely occur at the onset. Neustaedter[1] calls attention to the frequent occurrence of nasopharyngeal symptoms in the prodromal period. The patient may have sneezing spells and examination of the throat will show an anemic, glistening edematous condition of the mucous membrane, with a serous, frothy exudate upon it. He claims this is characteristic. At this time, or some days after, more or less general paralysis or paresis will be noticed. Most of the muscles, however, soon recover in a period varying from a few days to several months, leaving a permanent paralysis of one or two limbs, or perhaps only a single group of muscles. The constitutional symptoms soon disappear; indeed, in some cases they are so slight as to escape notice. Atrophy and flaccidity of the muscles that are to be permanently disabled soon appear, and in about a week's time examination will show the presence of the De R, which, as a rule, becomes typically developed.

Permanent paralysis most commonly affects the leg muscles, and the extensor groups more often than the flexors. The paralysis affects groups of muscles acting functionally together, and not groups that have the same nerve supply, as is the case in neuritis. Thus the biceps, brachialis anticus, and supinator longus, muscles which flex the forearm, are often paralyzed together.

The deep reflexes are abolished in the affected muscles. Sensory changes are absent. In severe cases retardation of the growth of the affected limb occurs, so that it is smaller than its fellow. Vasomotor changes are present, causing the affected parts to be cyanosed and feel cold to the touch. Owing to the want of support by the paralyzed muscles about them, the joints are relaxed and *dislocations* may occur. Also owing to the overcontraction of unantagonized muscles, deformities, such as various forms of *talipes*, may result. If the back muscles are involved, spinal curvature may

[1] New York Med. Jour., September 14, 1912, p. 519.

develop. While in most cases the onset is abrupt, there are instances in which it is gradual, taking several days for the disease to develop. Infrequently the bulbar nuclei of the cranial motor nerves are also affected, with consequent paralysis of the muscles supplied by them. (See Acute Bulbar Palsy, p. 264.)

Morbid Anatomy.—The primary action of the poison is upon the bloodvessels, the process therefore is interstitial instead of parenchymatous. Such being the case, the acute poliomyelitis is not a true system disease, but may be most conveniently so classified.

Changes are most frequently found in the lumbar segments. In the early stages the cord will be found redder and softer than normal. Minute hemorrhages may be present in the anterior horns and the bloodvessels are distended. In old cases the cord is smaller and denser. Upon section one or both of the anterior cornua are found reduced in size, and the anterior roots from the diseased region are shrunken and fibrous.

Microscopically, in the early stages the bloodvessels of the anterior cornua, septum, and commissure will be found distended and surrounded by a wall of round cells, and there will also be found an increase in the growth of neuroglia, due to irritation by the poisonous agent which has escaped from the blood. The ganglion cells are swollen and colorless, and show the evidences of degeneration, more or less marked according to the duration of the disease. The cells in the neighborhood of the vessels are more affected than those remote, as well as those groups of cells which have the same vascular supply. The fibers of the anterior roots show Wallerian degeneration, and the affected muscles fatty degeneration and atrophy of the fibers. In cases of long duration the ganglion cells in the affected area will be found to have more or less completely disappeared. Atrophy of the precentral convolutions and pyramidal tracts have also been found in cases of long standing. The parenchyma of the heart, liver, and kidneys is the seat of cloudy swelling and the lymph nodes and spleen show hyperplasia and proliferated endothelial cells.

Diagnosis.—Cases with severe constitutional disturbances may be mistaken for *meningitis;* the early appearance of paralysis and subsequent history of the disease should make the diagnosis clear. Lumbar puncture may be of assistance, especially in distinguishing from cerebrospinal meningitis, (p. 221), as in poliomyelitis the fluid is clear, apparently sterile, but contains Fehling's reducing substance, a large number of lymphocytes, and excess of globulin. These conditions are very similar to those found in tubercular meningitis (p. 227), so that unless tubercle bacilli are found the aid given in this disease is not great. The clinical symptoms of tubercular meningitis are usually characteristic enough to prevent a mistake. In poliomyelitis the condition of the fluid changes to apparently normal as soon as paralysis appears.[1] Examination of the throat (p. 258) may assist in making an early diagnosis, and if the appearance described is found, the cerebrospinal fluid should at once be examined. *Multiple neuritis* is distinguished by a progressive rather than a retrogressive course, such as occurs in poliomyelitis; by the presence of pain, tenderness over the nerve trunks, and often anesthesia; also the paralysis is always symmetrical.

Acute transverse myelitis is very rare in children, but may be mistaken in adults; the presence of anesthesia, symmetrical paralysis, sphincter paralysis, and bed-sores are characteristic of transverse myelitis, but not of poliomyelitis. The seat of the lesion in transverse myelitis is usually dorsal, in which event the deep reflexes would be increased and the muscles not wasted.

The *cerebral palsies* are distinguished by the absence of atrophy and electrical changes, and the frequent occurrence of mental symptoms (p. 250). The *pseudoparesis* of *rachitic children* is distinguished by the absence of atrophy and electrical changes, and the characteristic signs of that disorder.

Prognosis.—Death from involvement of respiratory centres sometimes occurs during the acute stage. After this is past

[1] Morse, Archives of Pediat., 1911, p. 164.

the prognosis as regards life is good. The electrical examination will help us in forming our prognosis as regards the ultimate return of power (p. 79). The possibility of the development of deformities and the possible retardation of growth must be remembered. Spinal disease as progressive spinal muscular atrophy or amyotrophic lateral sclerosis may develop in later years in those who have had acute poliomyelitis in childhood.[1]

Treatment.—If seen in the acute stage, perfect rest, preferably either on the side or prone; ice to the spine, laxatives, preferably calomel, full doses of hexamethylenamin and diaphoretics are proper. The patient should be isolated, the mouth and nose kept clean with antiseptic, alkaline douches, preferably containing formalin, and the excreta carefully disinfected (p. 257). The room should be fumigated after the patient is able to be about. After this stage has passed, usually in three weeks at the most, the use of electricity and massage should be begun (pp. 87 and 88). The affected limbs must be kept warm. Attempts to use the affected muscles must be encouraged. Tonics, of which strychnine in full doses occupies first place, should be given. Iron, phosphorus, cod-liver oil, and others may be given as circumstances require. If deformities or luxations occur, much can often be done by the proper application of braces, and in the case of contractures by tenotomies. Good results have been reported from the transplanting of the tendons of paralyzed muscles on to neighboring healthy muscles. And in suitable cases, *i. e.*, when only a few muscles are affected, from nerve anastomosis. Other orthopedic measures, as arthrodesis, may be valuable in proper cases.

Chronic Anterior Poliomyelitis.—Etiology.—This is a rare condition; it may follow acute poliomyelitis or possibly be due to a toxemia as lead.

Morbid Anatomy.—In the case reported by Spiller and Moleen[2] there was disappearance of many of the nerve cells of the anterior horns of the cord and of motor nuclei in the

[1] University of Pennsylvania Med. Bull., March, 1903.
[2] Amer. Jour. Med. Sci., December, 1905, p. 1025.

medulla, also disappearance of the anterior nerve roots and of the motor nerves of the medulla and numerous small hemorrhages in the gray matter, especially of the cord (Fig. 62).

FIG. 62

Showing the numerous hemorrhages within the gray matter of the spinal cord.

Symptoms.—The first symptom noticed is usually weakness in either the arms or legs, which progresses, or, as in the case reported by Spiller and Moleen,[1] there was a sudden paralysis of a group of muscles in the leg followed by wasting

[1] Loc. cit.

and De R. From this weakness and atrophy gradually developed in other muscles, until a year later there was involvement of the thenar and hypothenar eminences, the interosseii, and tongue. Fibrillary tremors are present in the muscles, and De R, either complete or incomplete, is present in the affected muscles. The deep reflexes in the affected parts are diminished or absent; in a few cases they have been increased. Slight pain may sometimes be present.

Diagnosis.—The distinction must be made between multiple neuritis, progressive spinal muscular atrophy, amyotrophic lateral sclerosis, and syringomyelia.

In the first, pain and tenderness over nerve trunks would usually be found, and fibrillary tremors absent. In neuritis due to lead, pain and tenderness are absent but other characteristic symptoms are present (pp. 128 and 131). In the second the symptoms (atrophy and weakness) usually begin in the hands (ulnar distribution), the atrophy is usually noticed before weakness, De R is not so marked until late in the disease. Single muscles are attacked at a time, while in poliomyelitis groups are affected (p. 269). The distinction, however, in many cases may be hard to make. The great increase in the deep reflexes, spasticity, and the Babinski sign will distinguish amyotrophic lateral sclerosis. In syringomyelia the sensory disturbances peculiar to that disease (p. 399) will be found.

Prognosis.—Complete recovery rarely, if ever, takes place. The disease may be arrested and some improvement occur. Sometimes the bulbar nuclei become involved and death results as in bulbar palsy (p. 265).

Treatment.—Rest in the early stages and avoidance of overexertion at any time. General tonics, nutritious food, and electricity and massage for the affected muscles.

Ophthalmoplegia.—Ophthalmoplegia, when due to degeneration of the nuclei, is a system disease of the peripheral motor neurone. This has been described on page 150.

Bulbar Palsy.—Bulbar palsy comprises disease of the nuclei of the motor cranial nerves from the seventh to the twelfth. Two *forms* are recognized—*i. e.*, acute and chronic progressive.

Acute Bulbar Palsy.—Etiology.—Acute bulbar paralysis may be due either to hemorrhage from or thrombosis occurring in the vessels supplying the pons and medulla (pp. 328 and 334) or to the influence of some toxic agent, in which it is cases analogous to acute anterior poliomyelitis. It is this form that is especially considered here. When the nuclei of the motor nerves of the eye are attacked the disease is known as *polioencephalitis superior*, and has been described on p. 150. When the nuclei from the seventh to the twelfth cranial nerves are involved it is often spoken of as *polioencephalitis inferior*.

In some instances the two may be combined. Infectious diseases appear to be a frequent cause. It may be associated with acute anterior poliomyelitis.

The **pathology** and **morbid anatomy** are practically the same as of acute anterior poliomyelitis.

Symptoms.—*Polioencephalitis inferior* usually begins with headache, vomiting, and vertigo. Difficulty in deglutition and articulation soon appear, and persistent hiccough may occur. The muscles of the lower part of the face, tongue, palate, and pharynx become rapidly paralyzed, and as a rule the patient soon dies from cardiac and respiratory failure. Consciousness is retained to the end. The disease may spread upward, causing paralysis of some or all of the ocular muscles (polioencephalitis superior); or downward, causing paralysis of the limbs (acute poliomyelitis).

Diagnosis.—Hemorrhage from or a thrombosis in the vascular supply of the medulla and pons is the only conditions that could be mistaken. In these the onset would be more sudden than in inflammation, and the paralysis would not likely be limited to the nuclei alone. There would probably be some immediate loss of power in the limbs and sensory symptoms, due to involvement of the motor and sensory tract as they pass to and from the cord (p. 334).

The **prognosis** is usually fatal.

The **treatment** consists of rest, strychnine, and other measures to support the heart and respiration.

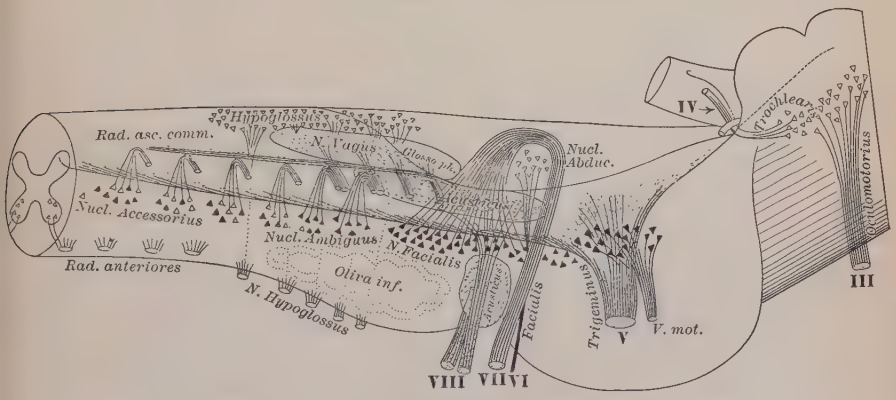
Chronic Progressive Bulbar Palsy. — Etiology. — This disease is also known as *glossolabiolaryngeal* paralysis. It

is a disease which occurs at the so-called degenerative period of life, between forty and fifty. The cause is not known.

Morbid Anatomy.—The symptoms are due to a symmetrical degeneration and atrophy of the ganglion cells comprising the motor nuclei (Fig. 63) of the seventh, ninth, tenth, and twelfth nerves; and rarely of the motor nerves of the eye.

Symptoms.—The first symptoms to attract attention are difficulties in articulation. Trouble with the linguals, as *l* and *r*, are first noticed; then trouble is experienced with

Fig. 63



Relative location of the nuclei of the different cranial nerves. (Edinger.)

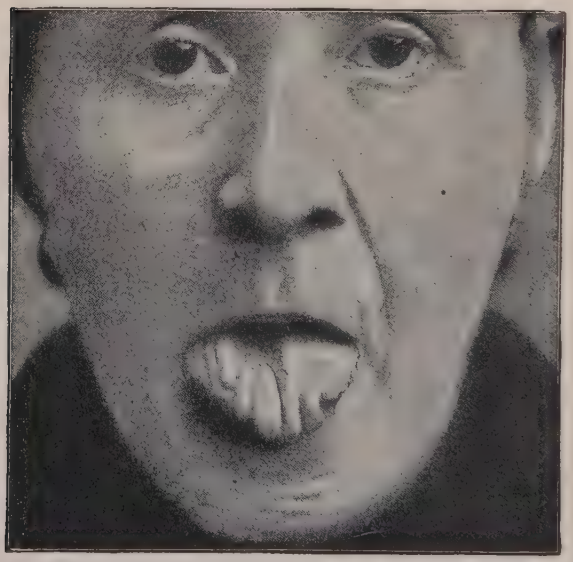
labials—viz., *b*, *p*, and *v*. Swallowing next becomes impaired and regurgitation of the food occurs, due to involvement of the pharyngeal and palatal muscles. The voice becomes nasal and vocalization more indistinct.

As the disease advances the muscles of the face become more involved, the lines become smoothed out, and expression is lost, due to their atrophy and paralysis. The tongue cannot be protruded and is noticeably atrophied (Fig. 64). The saliva drips from the sides of the mouth (drooling). Fibrillary tremors may be detected. Palatal, pharyngeal, and laryngeal reflexes disappear, but the jaw-jerk is fre-

quently present. As the disease progresses the cardiac and respiratory centres become involved and death results.

Rarely the ocular nuclei become involved. More frequently the disease extends down the cord, producing symptoms of amyotrophic lateral sclerosis or of chronic poliomyelitis (p. 261). The patient is emotional, laughing or crying without cause. There are no sensory symptoms.

FIG. 64



Atrophy of the tongue and lips in glossopharyngeal paralysis. (Oppenheim.)

Diagnosis.—The disease may be confounded in the early stages with the similar stage of *paretic dementia*, but in the former the absence of the peculiar mental and pupillary symptoms (Argyll-Robertson) and the appearance of difficulty in deglutition will soon decide the matter. Bilateral lesions confined to the lower extremities of the central convolutions, or internal capsules, may produce similar symptoms to those of bulbar palsy; such cases are known as

pseudobulbar palsies, and are distinguished by their frequently being asymmetrical in distribution and by a history of successive apoplectic attacks and non-progression of the symptoms unless another attack occurs, and by the absence of fibrillary tremors and atrophy of the tongue.

Myasthenia gravis or asthenic bulbar palsy may also be mistaken. This is described below. *Tumors of the medulla* may produce similar symptoms, but in such a case both sides would probably not be affected symmetrically, and the corticospinal sensory and motor tracts would also probably suffer.

Implication of the cranial nerves by a *basal meningitis* may be confounded with it; but in this the paralysis is not apt to be strictly symmetrical, and nerves not involved in bulbar palsy are often involved, as, for instance, the associated palsy of the sixth, seventh, and eighth. Headache, optic neuritis, and other symptoms of meningitis would be also present.

The **prognosis** is most grave. Few patients survive more than five years.

Treatment.—This consists of measures to improve the general health and nutrition. Physical and mental rest; concentrated, semisolid, and easily digested foods. The use of strychnine and other tonics. The use of mild galvanic currents. Artificial feeding is usually necessary.

Myasthenia Gravis Pseudoparalytica or Asthenic Bulbar Palsy.—Definition and Etiology.—While it cannot be said that this is a disease of the peripheral motor neurones it may conveniently be described here as the symptoms simulate those of chronic bulbar palsy. Other parts of the motor nervous system may also be involved. The disease usually occurs under thirty. Traumatism, syphilis, physical and mental exhaustion, and emotional strain have been given as causes. It is most probably due to the action of some unknown toxic agent. Pemberton[1] suggests that it is a disease of deranged muscular metabolism as indicated by marked calcium loss and reduction of creatinin output.

[1] Amer. Jour. Med. Sci., June, 1910, p. 816.

Morbid Anatomy.—No lesion of the nervous system has been found. Disease of the thymus gland has been present in several cases.[1]

Symptoms. The characteristic feature of the disease is an easy exhaustibility of the muscles, which is recovered from after a brief rest. Thus the patient will begin to walk in a normal manner, but after he has gone a short distance the legs will give out and he may fall. The levator palpebrarum muscle is very liable to be affected, so that after the eyes are held open for a few moments the lids will gradually droop and a double ptosis result. After a period of rest the muscles regain their power. All the muscles of the body may be affected, but those supplied by the cranial nerves are especially liable. The myasthenic reaction (p. 77) is a characteristic symptom, but may not always be present. Care must be taken not to excite the respiratory muscles in applying it, as death has resulted from their exhaustion. Attacks of dyspnea and tachycardia occur after exertion. The knee-jerks are present, but easily exhausted. Sensory changes are not present.

Diagnosis.—From chronic bulbar palsy the disorder is distinguished by the absence of drooling, fibrillary contractions, and atrophy of the affected muscles; and the presence of the peculiar transitory occurrence of the symptoms. The muscles supplied by the third motor division of the fifth and seventh nerves are more likely to be affected than in the organic affection. From pseudobulbar palsy we distinguish it by the presence in the former of permanent paralytic symptoms, of symptoms pointing to cerebral disturbance, headache, vertigo, mental confusion, etc.

The symptoms which distinguish disease of the nerves at the base from chronic bulbar palsy also obtain here. In addition, muscular atrophy and changes in the electrical reactions would be found. It may also be confounded with polioencephalitis, but in this disease the onset is different, and the symptoms do not improve to reappear after exertion.

Rarely tumor, brain syphilis, intermittent claudication

[1] Hun, Albany Medical Annals, January, 1904, p. 29.

(p. 499), and hysteria may produce similar symptoms. These would be distinguished by the presence of other symptoms of these disorders.

Prognosis.—Severe cases may cause death, but the patient may have a number of attacks, recovering either partially or completely from all. The disease lasts from two to four years.

Treatment.—Rest, careful feeding, artificial if required. Strychnine in full doses, calcium salts and other tonics and nutrients, as malt and cod-liver oil, should be given.

PROGRESSIVE SPINAL MUSCULAR ATROPHY (ARAN-DUCHENNE TYPE)

Etiology.—This disease usually begins in middle life, rarely before the twentieth year. Trauma, cold, and overexertion have been given as causes. Some cases are due to lead poisoning, others have been reported that are of syphilitic origin.[1] It is a rare disease. Werding and Hoffman have, however, described cases of this type beginning within the first month of life and which may affect a number of persons in the same family.

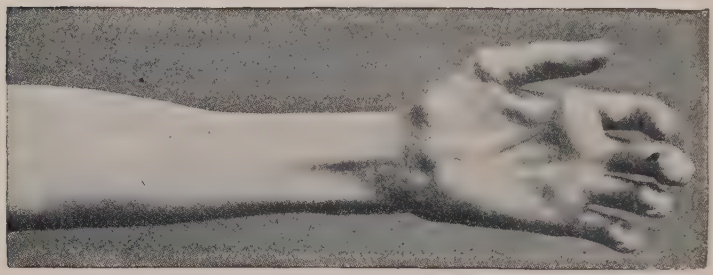
Morbid Anatomy.—Consists of atrophy and disappearance of the anterior horn cells, slight degeneration of the pyramidal and anterior lateral tracts may also be found. The muscles appear pale and yellowish red, and contain fatty stripes. Microscopically we find decrease in the size of the fibers, with degeneration of the muscle tissue to a fatty and granular mass.

Symptoms.—It develops insidiously and atrophy may exist for months before weakness is noticed. The atrophy usually begins in the small muscles of the hand (interosseii, opponens pollicis, adductor pollicis), causing the peculiar deformity known as "clawhand" or "main en griffe" (p. 192) (Figs. 65 and 65). But other muscles may be first affected (extensors of hand). After a time, or it may be the first symptom to

[1] Spiller, Jour. Nerv. and Ment. Dis., September, 1912, p. 584.

attract attention, weakness is noticed. Other muscles become affected, usually those of the shoulder girdle, forearm, and back. Fibrillary tremors are always present. The

FIG. 65



Hand and forearm in chronic spinal muscular atrophy, showing especially wasting of the thenar and hypothenar eminences and of the abductor indicis. (Dercum.)

FIG. 66

Atrophy and paralysis with main en griffe in chronic anterior poliomyelitis. (Starr.)

deep reflexes are diminished or lost. Electrical changes consist at first of a quantitative decrease; later De R develops. Sensory symptoms, excepting at times slight aching in the limbs, are absent. Bulbar nuclei may become affected.

Diagnosis.—It must be distinguished from chronic anterior poliomyelitis, amyotrophic lateral sclerosis, syringomyelia, arthritic atrophy, muscular dystrophies, progressive neuritic atrophy, cervical pachymeningitis, pressure from cervical ribs, and cervical caries. The first is distinguished by the fact that paralysis begins before atrophy develops (p. 262); the differential points between it and the second and third are given on page 286.

The primary occurrence of joint inflammation is characteristic of arthritic atrophy, and there is no progression (p. 273). Absence of fibrillary tremors and electrical changes, the distribution of the atrophy, and their hereditary nature distinguish the dystrophies (p. 276). In cervical pachymeningitis there is evidence of nerve-root irritation (p. 212) and in spinal caries pain, tenderness on jarring, deformity, etc., will be present. For the distinguishing points from progressive neuritic atrophy see page 274. A skiagram will show cervical ribs if present.

Prognosis.—The disease may continue for a number of years and may be arrested in its progress. Patients frequently die of some pulmonary condition or from involvement of the bulbar nuclei.

Treatment.—Consists of rest, forced feeding, general tonics, and nutrients and strychnine. Mild applications of electricity may be of service.

Acute Ascending Paralysis.—Definition and Etiology.—This disease, also known as *Landry's disease*, is a progressive paralysis, usually beginning in the legs, involving the trunk and arms, and finally the muscles of respiration, without marked sensory symptoms, and changes in electrical reaction. It usually occurs between the ages of twenty and thirty years. In a number of instances it has followed the infectious diseases, and is probably due to the action of some toxic agent.

Morbid Anatomy.—A number of different anatomical conditions have been found in cases in which a diagnosis of Landry's disease had been made. For instance, multiple neuritis, poliomyelitis, and acute diffuse myelitis. The symptoms are due to the action of a poison upon the peripheral motor neurone. Many of the earlier cases showed no

pathological changes whatever, but with improved methods of staining more or less marked changes in the ganglion cells of the anterior horns have been found. In some cases these have been associated with dilatation of the bloodvessels and perivascular infiltration, as in acute poliomyelitis. In others there has been parenchymatous degeneration of these cells. Associated with these cell changes degeneration of the peripheral nerves is found. Mills and Spiller believe that in addition to changes such as above described, Landry's paralysis may be due to a diffuse myelitis. Cases no doubt occur in which the toxemia is so malignant that death occurs before anatomical changes develop. In one case a micrococcus resembling that of meningitis was found.

Symptoms.—Weakness of the legs is the first symptom, which progressively grows worse, and often with great rapidity. The muscles of the trunk next suffer, and in a few days, or even less, the arms are affected. The muscles of the neck next suffer, and finally those of articulation, deglutition, and respiration. The reflexes are lost, but the muscles do not waste nor show electrical changes. The sphincters usually are not involved. Sensory symptoms are, as a rule, absent, but hyperesthesia and various paresthetic sensations have been noted in some cases. Rarely the disease may begin above and *descend*. Enlargement of the spleen has been noted.

Diagnosis.—The diagnosis must be made from multiple neuritis, acute poliomyelitis, and acute myelitis. From the first the disease differs in the absence of marked sensory symptoms, muscular atrophy, and changes in the electrical reactions. From the second it differs in its progressive ascending course and in the absence of muscular atrophy and changes in the electrical reactions. From the third, in the absence of anesthesia, bed-sores, and sphincter paralysis.

Prognosis.—Prognosis is usually fatal. Death has occurred in forty-eight hours. Other cases may persist for a week or two. Recoveries have been reported.

Treatment.—The treatment consists of rest and supporting measures. Dana recommends warm baths or packs, counter-

irritation of the spine, and laxatives. Large doses of ergot have been recommended. Salicylates, hexamethylenamin, iodide of potassium, and mercury may be tried.

Arthritic Muscular Atrophy.—Definition.—An atrophy and weakness occurring in the muscles about diseased joints, which bear no relation to the nature, cause, or severity of the joint affection.

Etiology. — Any joint inflammation — *i. e.,* traumatic, rheumatic, gonorrheal, or other form of arthritis—may cause it.

Pathology.—The muscle fibers are not degenerated, but are narrowed, paler, and more flaccid. The nuclei may be increased. The condition is probably a reflex one, dependent upon irritation of the articular nerve ending, which is centripetally propagated to the cells of the anterior horns which give origin to the motor nerves. The change in these cells is a molecular one.

Symptoms.—Either during the acute symptoms in the joint or after their subsidence atrophy is noticed in the muscles surrounding it. The extensors of and muscles to the proximal side of the joint are principally and may be the only ones affected. Thus, if the shoulder-joint is diseased, the deltoid, triceps, and scapular muscles are the usual ones involved. Along with the atrophy, weakness of the muscles will be observed. The electrical reactions are normal or quantitatively decreased. Mechanical irritability may be increased in the affected muscles, so that if the thigh muscles are affected the knee-jerk is increased. Fibrillary tremors are often present. Sensory symptoms are absent.

Diagnosis.—The occurrence of atrophy following inflammation in a joint is characteristic of this condition. From nerve injury following dislocation the arthritic atrophy is distinguished by the history, the absence of changes in the electrical reactions and of nerve tenderness. Changes and adhesions in joints sometimes occur secondarily to neuritis; in these, however, the atrophy precedes the joint trouble, and other evidences of neuritis are present. The early stage of progressive muscular atrophy might also be confounded with it. In such a case it might be necessary to await for

a time the development of the disease before giving a positive opinion.

Treatment.—The treatment consists in the relief of the inflammation of the joint and the employment of electricity, massage, and strychnine.

Progressive Neuritic Muscular Atrophy.—Etiology.—This form of atrophy, also known as either the peroneal type of progressive muscular atrophy, Charcot-Marie-Tooth form of progressive muscular atrophy, progressive neurotic atrophy, or progressive neural muscular atrophy of Hoffman is a form of atrophy beginning in the intrinsic muscles of the feet and extending upward. It may make its appearance at any period between infancy and late in life, but usually does so before the twentieth year. It is more common in males than females. It is an hereditary or family disease; no other cause is known.

Morbid Anatomy.—There have been found degeneration of the nerve fibers, excess of connective tissue with proliferation of cells in the neurilemma; usually also degeneration of the posterior roots, with degeneration in the posterior columns, especially those of Burdach, slight degeneration of the pyramidal tracts and columns of Clarke, and changes in the ganglion cells of the gray matter. The muscles show atrophy of their fibers with proliferation of connective tissue.

Symptoms.—The small muscles of the foot are usually attacked first, then the disease spreads to the peronei, extensor longus digitorum, and tibialis anticus. The weakening of these muscles causes club-foot, at first talipes cavus, which later develops into pes equinus or equinovarus. The muscles of the calf then suffer, then those of the thigh, especially the vastus internus. About this time the small muscles of the hand become atrophied, as in progressive muscular atrophy, then the forearm muscles (Fig. 67). The muscles of the upper arm, neck, face, and body but rarely become affected. Fibrillary twitchings are seen in the affected muscles. The deep reflexes are abolished as the disease advances. Changes in the electrical reactions occur early —first a quantitative decrease, then a De R being found. Vague pains and paresthesiæ are often present, and dimin-

ished sensibility is found in some cases. The affected limbs are apt to be cyanotic and cold. Both limbs are, in most cases, affected, but the process may be confined to one. Very rarely it may begin in the hands. Cases have been reported in which with the symptoms above described there has been optic atrophy, mental impairment, and paralysis of cranial nerves.[1] Cataract seems to be also a rare complication[2] (see p. 284).

Diagnosis.—The disease may be confounded with progressive muscular atrophy, multiple neuritis, and muscular dystrophy. From the first it differs in the mode of onset, the age of the patient, and possible existence of a history of heredity.

From multiple neuritis it differs in its mode of onset, method of progression, and the absence of nerve tenderness. In the muscular dystrophies, fibrillary tremors, marked changes in electrical reac-

FIG. 67

Charcot-Marie-Tooth disease. Atrophy of the legs and drop-feet, and atrophy of the hands. (Starr.)

[1] Bertolotti, Abstracted in Rev. Neurol. and Psychiat., August, 1910, p. 497.
[2] Hamilton, Rev. Neurol. and Psychiat., December, 1911, p. 645.

tions, and sensory symptoms are not found. Rare cases of dystrophy occur in which the atrophy begins in the hands and feet. The differential diagnosis in these cases may be very difficult.[1]

The **prognosis** as regards cure is bad, but the progress may be slow.

Treatment.—Treatment consists of tonics, and the use of electricity and massage to promote the nutrition of the muscles. Resulting deformities may be helped by tenotomies and braces.

The Muscular Dystrophies.—Definition.—While probably due to disease of the muscles themselves they may be conveniently discussed here for purposes of contrast. They are hereditary affections characterized by progressive muscular wasting beginning in certain groups of muscles, which is sometimes preceded by apparent hypertrophy; and sometimes associated with apparent hypertrophy of other muscles. They are also known as *myopathies.*

Etiology.—The only etiological factor known is the influence of heredity, the disease occurring in several generations. The disease usually sets in before puberty, but may appear later.

Morbid Anatomy.—In the early stages true hypertrophy of muscle fibers may be observed. In others there is proliferation of muscle nuclei and longitudinal splitting of the fiber; along with this there is increase of connective tissue, which takes the place of the destroyed muscle fibers. In the pseudohypertrophic form there is often a deposit of fat in the connective-tissue cells, which increases until extensive lipomatosis results. Later this may be absorbed. Evidences of degeneration have been found in the cord and nerves, but usually they are normal in appearance.

Symptoms.—A number of different clinical types of muscular dystrophies have been described dependent upon the age at the onset, muscles first affected, the occurrence of hypertrophy, etc. They are:

I. Pseudomuscular hypertrophy of Duchenne.

[1] Spiller, Jour. Nerv. and Ment. Dis., January, 1907, p. 14.

II. Landouzy-Dejerine type, or infantile progressive muscular atrophy of Duchenne, or facioscapulohumeral type

FIG. 68

Pseudomuscular hypertrophy in brothers (Infirmary for Nervous Diseases, Philadelphia).

which appears in young children; the facial muscles are first involved.

FIG. 69

Pseudohypertrophic paralysis. The act of rising. The child rolls over on the face. (Starr.)

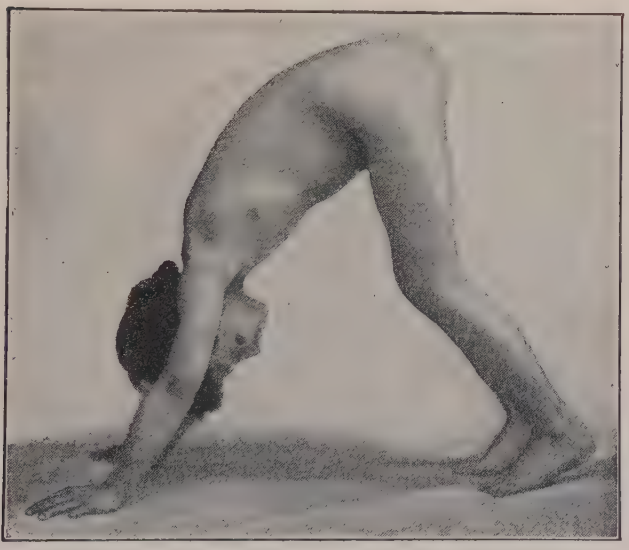
FIG. 70

Pseudohypertrophic paralysis. The act of rising. The child raises the trunk on hands and knees. This position shows the weakness of the muscles of the neck and the atrophy of the arms. (Starr.)

III. Erb's juvenile type or scapulohumeral type, appearing in late childhood or youth, and beginning in the muscles about the shoulder.

There is no sharp line of division between these types. Each of the different types may occur in the same family, and the peculiar characteristics of one type may be present in another. In all of these forms sensory symptoms are

FIG. 71



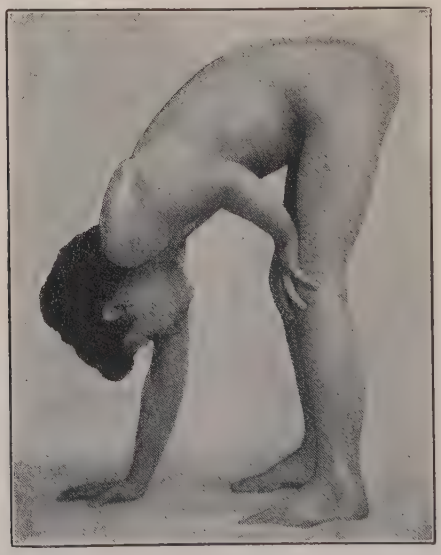
Pseudohypertrophic paralysis. The act of rising. The child raises the trunk by bringing the feet forward and extending the legs at the knee. (Starr.)

absent; there are no fibrillary tremors; the De R is usually absent, and the deep reflexes that are connected with affected muscles are absent. The parts are cold and cyanosed, and the intelligence is good. Deformities often develop. The peculiarities of each type will be described separately.

Pseudomuscular Hypertrophy.—This form usually appears under the age of ten. Weakness is first noticed in the legs, shown by clumsiness and a tendency to fall, and a waddling

gait. Difficulty in rising when seated on the floor to an erect position is an early symptom. The patient accomplishes this by first getting into a position similar to that assumed in playing leap-frog. He then raises the body by climbing up his legs with his hands (Figs. 69 to 73). Shortly after this the calves will be found to feel firm to the touch. The hypertrophy may not extend beyond this, or it may increase and the legs assume huge proportions. The extensors of the

FIG. 72



Pseudohypertrophic paralysis. The act of rising. The child raises the shoulders by supporting the weight on the hand pressed upon the knee. (Starr.)

leg—glutei, lumbar muscles, deltoids, triceps, and infraspinati, in the order given—next become hypertrophied. The attitude when erect is characteristic: the legs are far apart, the back curved, the shoulders thrown back, and the abdomen protruded (Fig. 68). The latissimus dorsi, lower part of the pectorals, upper arm, and thigh muscles usually become atrophied. After a time the hypertrophy may disappear and be succeeded by atrophy.

Facioscapulohumeral Type.—This type begins usually about the third or fourth year. Atrophy first attacks the face, giving it a peculiar appearance; weakness of the orbicularis oris causes the lips to protrude, producing the so-called "tapir mouth." The eye muscles and those of mastication

FIG. 73

Pseudohypertrophic paralysis. The act of rising. (Starr.)

and deglutition are not involved. The atrophy then extends to the shoulders and arms, and the subsequent course is as in other types (Fig. 74).

Scapulohumeral or Erb's Type.—This type usually appears later in life than the two preceding types. The muscles of the shoulder girdle are first attacked; then those of the arm, back, part of the pectorals, latissimus dorsi,

trapezii, and, finally, the thigh muscles. The forearm and legs below the knee are involved very late, if at all.

FIG. 74

Progressive muscular dystrophy, atrophic type of Landouzy-Dejerine. Age, nine years; duration, seven years. Face, arms, and legs extensively atrophied. Face expressionless; pectorals and deltoids wholly atrophic; thighs atrophied. The forearms and the legs are normal. (Curschmann.)

The supraspinati and infraspinati also usually escape (Fig. 75). Rarely the atrophy may begin in the hands and feet (p. 276).

Diagnosis. — The peculiar features above described should usually enable a diagnosis to be made without difficulty (p. 279). Progressive muscular atrophy, progressive neuritic atrophy, and multiple neuritis are the only affections that could be confounded with them.

Prognosis.—There is no cure known. Their progress, however, is usually slow, and the patients may live twenty-five to thirty years.

Treatment.—A mother who is a member of a dystrophic family should not suckle her children. Infants born of such parents should have the best of hygienic care and surroundings. Tonics of all sorts, especially strychnine, are indicated. Massage and electricity may be employed. Much benefit has been derived from systematic but gentle gymnastic exercises. For the contractures which may result tenotomies may prove useful.

Myotonia Atrophica.—This rare condition may be mistaken for myopathy and is characterized by weakness of the facial muscles causing an appearance resembling the

FIG. 75

Erb's juvenile dystrophy. The disease began at the age of sixteen; photograph made at the age of thirty. Supra- and infraspinati and gastrocnemii hypertrophied. Trapezii, deltoids, biceps, triceps, latissimi dorsi, serrati magni, erectores spinæ, all muscles of thighs, and glutei are atrophied. (By permission of Dr. G. L. Walton.)

facioscapulohumeral type of myopathy, atrophy of the sternomastoids, of the vasti of the thighs and dorsal flexors of the feet and *a slow* relaxation of certain muscles after contraction. The more forcible the contraction the slower the relaxation.[1] Other muscles than those mentioned may be atrophied. It is in some instances a familial disease and cases have been reported in which cataract also occurred early in life.[2] (See p. 275.)

DISEASES AFFECTING BOTH CENTRAL AND PERIPHERAL MOTOR NEURONES

Amyotrophic Lateral Sclerosis.—Etiology.—The disease is more common in males than females, and usually occurs between the ages of thirty and fifty. Cases may occur on either side of these limits. It is probably an early death of the motor system due to inherent weakness (abiotrophy).

Exposure to cold and wet, mental strain, infectious diseases, acute rheumatism, and syphilis have been mentioned as causes. Chronic lead poisoning is a cause, possibly also other forms of toxemia may be etiological factors.[3] The disease is not hereditary.

Morbid Anatomy.—The principal anatomical changes are a slow degeneration of the cells in the anterior horns of the cord; and, if bulbar symptoms are present, in the nuclei of the motor cranial nerves, the degeneration usually commences in the cells situated in the lower cervical and upper dorsal regions, and is accompanied by increase of the neuroglia and degeneration of the fibers forming the peripheral nerves and their intramuscular endings; degeneration and consequent atrophy of the muscle fibers, more or less degeneration of the pyramidal tracts extending toward the brain, or the entire tract from the cortical cells downward may be involved. The anterolateral columns in the vicinity of the anterior horns may be also affected.

[1] Batten and Gibb, Brain, 1909, p. 187.
[2] Greenfield, Rev. Neurol. and Psychiat., April, 1911, p. 169.
[3] Wilson, Review of Neurology and Psychiatry, June, 1907, p. 441.

Symptoms.—The patient usually first suffers for a time from rheumatoid pains in the shoulder and hand. A feeling of numbness and weariness may also be complained of. Muscular wasting is usually noticed in one hand, especially in the muscles supplied by the ulnar nerve, the adductor pollicis being usually first affected. Other muscles of the ball of the thumb, the lumbricales, and interossei soon follow, leaving depressions between the metacarpal bones. As the atrophy becomes more intense the clawhand described on page 269 is produced (Fig. 65). The atrophy gradually extends up the arm to the shoulder, during which time a period of from three to nine months, atrophy will probably also have begun in the other hand. If the disease continues to spread, the muscles of the back and intercostals are next attacked; then the muscles of the hip and thigh, the glutei, abductors, and extensors of the leg being most often affected. The muscles below the knee may be next involved, but they usually escape. Sometimes the process begins in the muscles about the shoulder (upper-arm type) instead of in the hand, to which it afterward extends or it may involve bulbar nuclei first. The disease may also ascend, causing paralysis of the diaphragm, and finally reaching the nuclei in the medulla (bulbar palsy). Cases also occur in which the symptoms for some time are unilateral.

The muscles as they atrophy become weak; but atrophy is noticed before the weakness, and weakness does not precede it, as in diseases of the nerve trunks and acute poliomyelitis. Fibrillary contractions of the muscles are marked, even in muscles that are not yet wasted. Excepting late in the disease, the De R cannot be obtained; but there is quantitative decrease in the response of the muscles to both the faradic and galvanic currents (p. 77).

There is no anesthesia, but the dull pains and paresthesia, which are present early, may continue.

Vasomotor disturbance, shown by excessive sweating and congestion, may be present in the affected parts. The sphincters are not attacked.

Owing to the lack of muscle support *deformities* may occur—viz., lordosis, due to weakness of the back muscles.

The head cannot be held erect from lack of support, and contractures and partial dislocations are observed.

Soon after the appearance of atrophy, or, indeed, in some cases it is noticed first, the legs become more or less spastic, the deep reflexes increased, and the Babinski sign is present. The atrophy may be confined to the intrinsic hand muscles, while the legs present the picture of a lateral sclerosis (p. 245). In other words, the disease in brief may be described as progressive spinal muscular atrophy plus lateral sclerosis. In the final stage dementia may occur.

Diagnosis.—*Amyotrophic lateral sclerosis* can be distinguished from *primary spastic paralysis* by the muscular wasting and the fact that it begins either in the arms or with bulbar symptoms. From *progressive spinal muscular atrophy* and *chronic anterior poliomyelitis* by the spastic condition of the legs, increased deep reflexes and Babinski sign. From *syringomyelia* by the occurrence in that disorder of the characteristic sensory disturbances (p. 49). From *chronic cervical pachymeningitis* (p. 212) by the absence of pain and evidence of irritation of the nerve roots. From *transverse myelitis*, by the absence of anesthesia or involvement of the sphincters.

Prognosis.—The prognosis as regards recovery is bad, but the disease may last for years before death ensues from bulbar involvement or lung disease. Remissions may occur, and sometimes its progress is permanently arrested.

Treatment.—This consists of rest, quiet, plenty of fresh air, and good feeding. Arsenic, phosphorus, cod-liver oil, and other tonics are indicated. Electricity, in the form of galvanization, and the cathode applied labile to the muscles, may be of service.

COMBINED SYSTEM DISEASES

When the disease affects *both afferent* and *efferent systems* of *neurones* it is called a *combined system disease*. While it is convenient to describe under this head the diseases which follow below, it should be remembered that it is denied by some that these are true system diseases, but are diffuse processes; also that in locomotor ataxia lesions of the motor

neurones may occur, and that in diseases primarily affecting motor neurones changes may be found in the posterior columns. This is the exception and not the rule. In the diseases about to be described the symptoms from the first point to involvement of both motor and sensory neurones.

Ataxic Paraplegia (Posterolateral Sclerosis, Progressive Spastic Ataxia).—Definition.—This is a condition presenting a combination of the symptoms of spastic paraplegia and locomotor ataxia.

Etiology.—The symptoms are due to degeneration of the posterior and lateral columns. While Gowers described what he believed to be a primary degeneration of these tracts, in most cases showing the symptoms it is secondary, due either to a previous transverse myelitis (p. 381) in which ascending and descending degenerations have occurred; vascular lesions (usually syphilitic) causing softening of the posterior portions of the lateral columns, and posterior columns (p. 410); toxic conditions (p. 292), Friedreich's ataxia (p. 288); or paresis (p. 415).

Morbid Anatomy.—Is that of the causes mentioned under etiology.

Symptoms.—The gait is a combination of spasticity and ataxia (pp. 55 and 246). Difficulty may be experienced in walking in the dark. The Romberg symptom is present, and the tests employed to detect incoördination (p. 55) reveal its presence in the legs and at times also in the arms. The gait is different from that of tabes, being equally unsteady, but the feet are neither raised so high nor do they descend to the gound so suddenly as is seen in that disease. Muscle sense is usually affected as in tabes.

The skin reflexes may be increased, normal, or diminished. The knee-jerk is much increased and ankle clonus can frequently be obtained. The Babinski sign is present. When the arms are affected a similar condition of reflexes is observed. Other symptoms may be present dependent upon the primary disease present (*vide*).

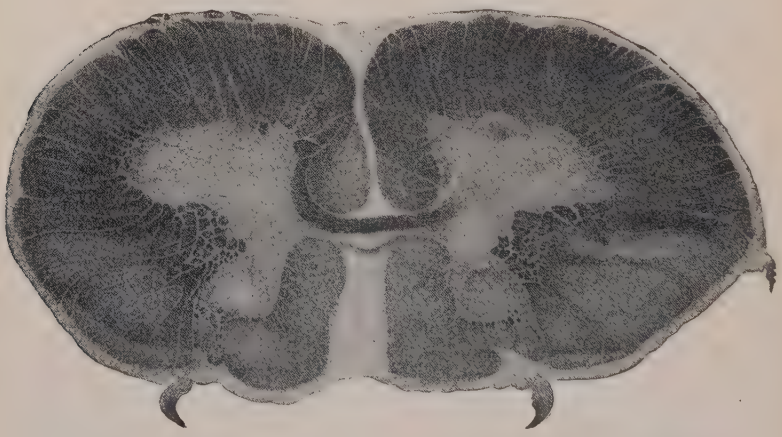
Diagnosis.—The disease can be distinguished from *tabes* by the preservation of the knee-jerks, existence of ankle clonus, difference in gait, and absence of lightning pains and

eye symptoms; from *spastic paraplegia*, by the unsteadiness of the gait.

In *cerebellar tumors* some or all of the general symptoms of tumor—*i. e.*, optic neuritis, nausea, headache, etc.—would be present. The distinctive symptoms of the various diseases causing the symptom-complex will be found under their various headings. (See Etiology.)

Hereditary Ataxia.—This affection may be divided into two types—*i. e.*, Friedreich's disease, in which the lesions begin and are most marked in the *cord;* and hereditary cerebellar ataxia, in which the *cerebellum* is first involved.

Fig. 76



The lesion of Friedreich's hereditary ataxia. Maldevelopment and sclerosis of the lateral and posterior columns. (Schultze, Lehrbk. d. Nervenkr., Taf. iv.)

Friedreich's Ataxia.—Etiology.—This disease, also known as Friedreich's disease, is a family disease, as many as ten cases in three generations of a single family having been reported. The parents may be perfectly healthy, but usually there is some degenerative tendency in some of the ancestors, as alcoholism, epilepsy, chorea, and syphilis. Isolated cases occur. The symptoms frequently appear after some infectious disease. The symptoms usually develop between the ages of six and fifteen.

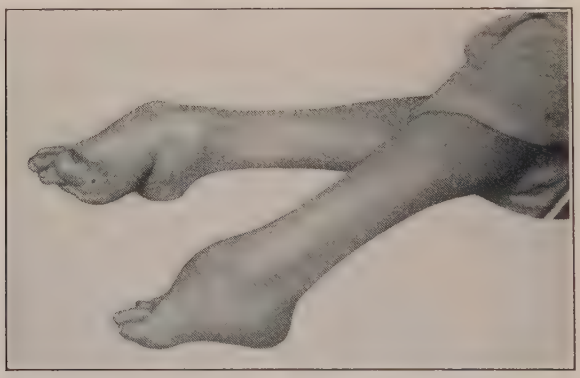
Morbid Anatomy.—The changes found consist of sclerosis of the posterior and lateral columns of the cord (Fig. 76). The posterior roots are also involved. The column of Goll is involved more than that of Burdach, in the root zone of which and in the column of Lissauer normal fibers are found. The direct cerebellar tracts and columns of Gowers also suffer. Changes in the ganglion cells of the anterior and posterior horns, and of the column of Clarke, and atrophic changes in the postpyramidal nuclei and restiform bodies have also been observed. Lesions of the cerebellum have been reported in a few cases. The change in this disease is a neuroglia sclerosis (gliosis) and not the ordinary connective-tissue sclerosis. It is probable that the disorder is a manifestation of a lower vitality than normal of the affected parts, so that they degenerate early in life (abiotrophy).

Symptoms.—Usually the first symptom noticed is a gradually developing incoördination, first in the legs and later in the arms. The gait is swaying, and resembles the gait due to cerebellar disease more than that of tabes (p. 55). In standing the legs are kept wide apart; the Romberg symptom is usually present, but may be absent. Movements of the arms are choreiform and irregular. In making voluntary movements the action is overdone, and when attempting to grasp an object the hand is held over it, moving about if for a moment and then pouncing upon it as an eagle upon its prey ("hovering hand"). In many cases, when the body is erect or the limb extended, there are irregular swaying, oscillating movements of the head, trunk, and extended limb (static ataxia). After a while the speech is affected, becoming slurring, with a hesitancy in the enunciation of sentences. The knee-jerks are in most cases lost early. The skin reflexes are normal. The Babinski reflex may be absent in the early stages but is present in advanced cases. Atrophy of the optic nerve and paralysis of the ocular muscles do not occur in this type (p. 291), but *nystagmus*, developed usually by movement of the eyes upward or laterally, is present.

Sensory symptoms seldom occur, and trophic lesions are rare. Involvement of the sphincters and mental symptoms

are also absent until late in the course of the disease. The face early becomes dull and expressionless and the mouth is often kept partly open. In the later stages weakness increases, some wasting of the muscles may occur, and either pes cavus, talipes equinus or varus and lateral spinal curvature develop. Flexion of the distal and extension of the proximal phalanx of the great toe is a characteristic symptom (Fig. 77).

FIG. 77



Friedreich's ataxia, showing the typical deformity of the feet.

Diagnosis.—The features which distinguish this from the *cerebellar type* will be made evident by a study of the symptoms of that form (p. 291).

From *locomotor ataxia* it differs in the early onset, absence of pupillary symptoms, presence of nystagmus and speech defects; muscular weakness, deformities, and the differences in the gait. Posterolateral sclerosis is distinguished by increased knee-jerks and absence of nystagmus (p. 287).

In *disseminated sclerosis* the knee-jerks are increased; the speech of a sing-song or scanning type, instead of hesitating; there are often optic atrophy and paralysis of ocular muscles; and it is not a family disease. In some cases, however, the diagnosis may be difficult. In *cerebellar tumor* the general symptoms of brain tumor are present, and choreiform and ataxic movements of the arms do not occur. From *chorea*

it is distinguished by its progressive character, absence of knee-jerks, absence of affection of the face, and the difference in the character of the movements. *Huntingdon's chorea*, while a family disease, begins in adult life, and mental symptoms soon appear.

Prognosis.—The affection is incurable, but lasts for many years, death occurring from an intercurrent disorder.

Treatment.—The treatment is symptomatic, and consists principally of tonics, nutrients, and good hygiene. Either arsenic or nitrate of silver may be tried. If the inheritance is from the mother's side, children should not be nursed by her.

Hereditary Cerebellar Ataxia (of Marie and Nonne).—**Etiology.**—The etiology is similar to that of Friedreich's ataxia.

Morbid Anatomy.—Atrophy of the cerebelium has been found in some cases. In others there were no changes visible to the naked eye; but by the use of the microscope the cells of Purkinje were found atrophied. In a case reported by Sanger Brown and Myer no changes were found in the cerebellum, but the tracts in the cord connected with cerebellar functions showed a condition of defective development.

Symptoms.—The family history, cerebellar gait, Romberg's symptom, tremor of the head and extremities, choreiform, awkward movements of the arms and hands, disordered speech, and nystagmus are symptoms common to both this type and Friedreich's. It differs in the usually later onset (puberty or later), the presence of increased or normal knee-jerks, occasional occurrence of ankle clonus and the frequent occurrence of ocular symptoms, viz., Argyll-Robertson pupil, diplopia, color blindness, and atrophy of the optic nerve. There may also be disturbances of sensation. Talipes and scoliosis rarely occur in this type. Mental deterioration sometimes develops. Purves Stewart[1] has reported three cases occurring in one family in which, with symptoms of ataxia paraplegia, early atrophy of the optic nerve and

[1] Rev. Neurol. and Psychiat., August, 1912, p. 357.

blindness occurred. He called them *familial amaurotic* ataxic paraplegia.

Diagnosis, Prognosis, and Treatment.—These are practically the same as those of Friedreich's type. In this form, however, the distinction from disseminated sclerosis is often very difficult.

Combined Sclerosis of the Spinal Cord (Putnam's Type and that of Pernicious Anemia).—Etiology.—This affection, also termed *subacute combined sclerosis* by Russell, Batten, and Collier, and *diffuse degeneration* of the spinal cord by Putnam and Taylor, occurs in persons past middle life, and is associated sometimes with pernicious anemia, but more commonly the anemia is secondary, it and the sclerosis being due to a toxemic condition, as influenza, chronic diarrhea, lead poisoning, pellagra (p. 537), and malaria. There is frequently a previous neuropathic personal or family history. Cases developing in the course of anemia were first described by Lichtheim in 1887; those produced by other causes, by Putnam, and a little later by Dana in 1891.

Morbid Anatomy.—The posterior columns are first and most involved, usually more intensely, in the cervical and dorsal regions; in some of the cases associated with pernicious anemia these were the only parts involved. In most cases the lateral columns, especially the crossed pyramidal tracts, are also affected. The other columns may be involved, but neither so constantly nor completely. The sclerosis may occur in focal areas not symmetrically distributed, which resembles multiple sclerosis in not causing secondary degeneration. Late in the course of the disease the anterior horns are attacked. There may be softening of the cord with the production of cavities. The bloodvessels are affected with hyaline degeneration.

The nature of the process is a primary nerve-fiber degeneration due to the action of the poison.

Symptoms.—The initial symptom is a persistent paresthesia, most commonly of the feet (with this there is some weakness), and a little later ataxia. Pain in the back and limbs may be present at this time. At first there are increased knee-jerks, ankle clonus, and some spasticity of the muscles.

Late in the disease the rigidity may disappear and the knee-jerks be lost. After a few months the arms become affected, the symptoms developing similarly to those of the legs. Rarely the trouble begins in the arms. Loss of tactile pain and thermic senses may be a late symptom; or there may be dissociation of sensation, as in syringomyelia. Atrophy of muscles may also occur. The sphincters are usually intact until near the end. A slight grade of dementia may develop.

Diagnosis.—The disease is distinguished from *locomotor ataxia* by its rapid onset, motor weakness, absence of ocular symptoms, increased knee-jerks (early stage), and the coexisting anemia.

From *ataxic paraplegia*, due to other causes (p. 287), by its rapid development and progress, the greater degree of weakness, and constitutional symptoms.

From *multiple neuritis*, by the absence of pain, tenderness, and marked muscular atrophy. Those cases in which dissociation of sensation occurs may be mistaken for *syringomyelia*, but the rapid development of the symptoms will in a short while distinguish the two.

Prognosis.—The prognosis is not good. Death may occur in from six months to three years. Some cases recover.

Treatment.—The treatment consists of arsenic, iron, quinine, and other tonics and nutrients.

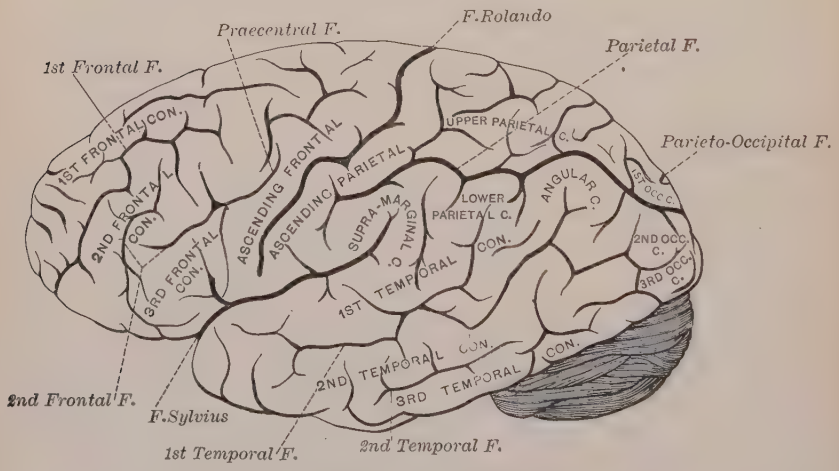
CHAPTER XI

DIFFUSE AND FOCAL DISEASES OF THE BRAIN

CEREBRAL LOCALIZATION

Reference to Figs. 78 to 81 will indicate the location and names of the various convolutions, sulci, and lobes of the brain, a knowledge of which is essential to the location of a lesion involving the cortical centres.

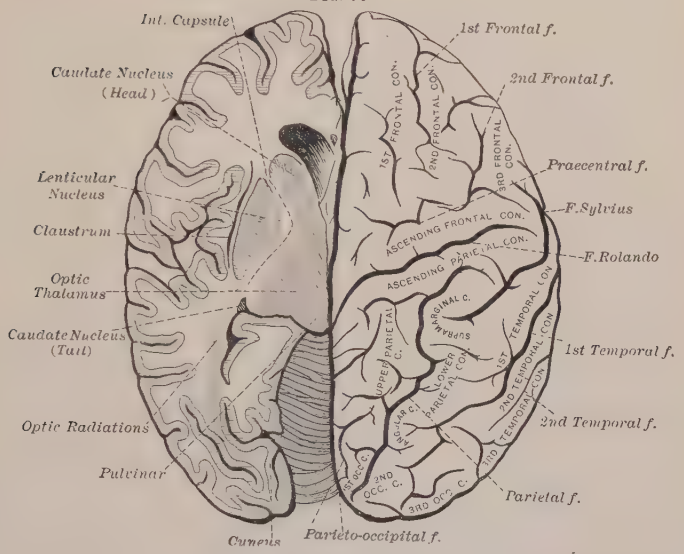
Fig. 78



Convolutions of the left hemisphere.

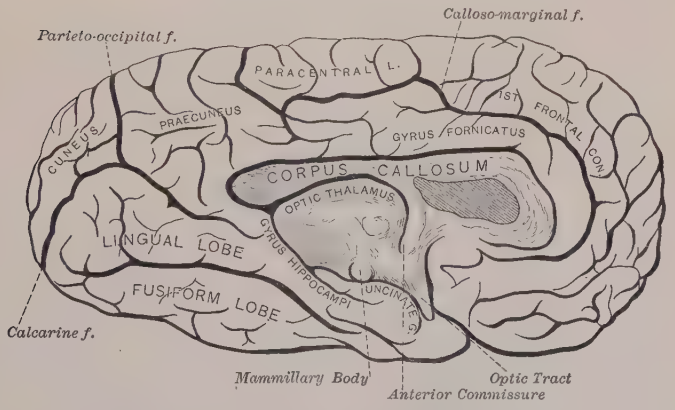
In determining the locality of a lesion in the brain it must be remembered that symptoms referable to the centres in which the lesion is actually situated are not the only symptoms that may be present, but that others, either irritative or paralytic, are usually present, due to irritation or compression

FIG. 79



Convolutions of the vertex, on the right; on the left, the basal ganglia, internal capsule, centrum ovale, and cuneus.

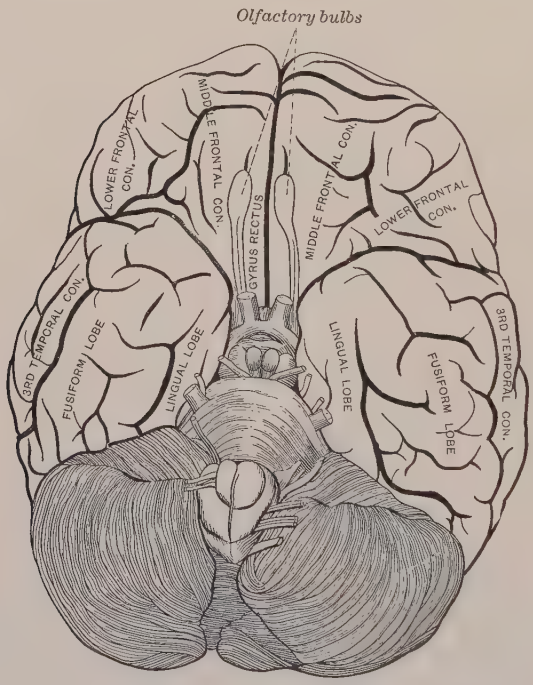
FIG. 80



Vertical section through the centre of the corpus callosum, showing the convolutions of the inturned surfaces of the hemispheres.

of neighboring centres or tracts. Also, that cortical centres represent movements and not muscles, therefore, several muscles may be represented in a centre. Some of the facts regarding cerebral localization are still in dispute, but the foregoing, it is believed, represents the most generally

FIG. 81



Convolutions of the base of the cerebrum and the cerebellum.

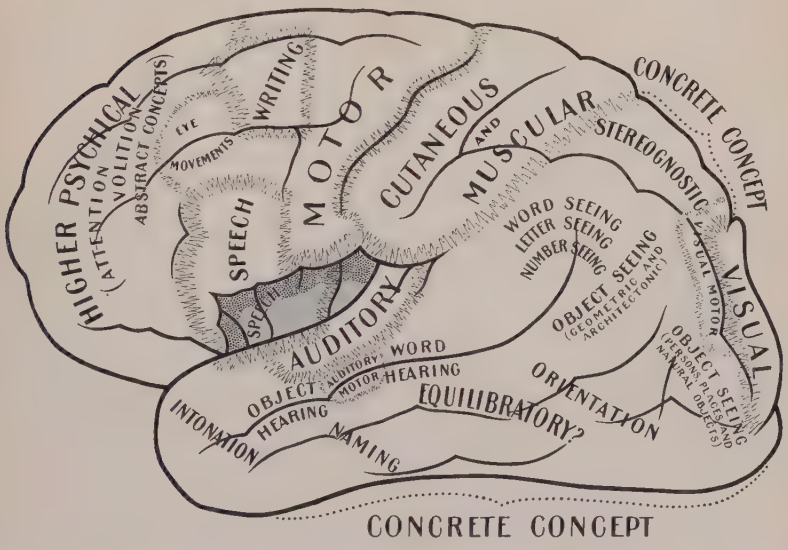
accepted views at this time. Figs. 82 and 83, devised by Dr. C. K. Mills,[1] represent the different areas of the cortex, and Fig. 84 represents the subdivisions of the motor area. The

[1] Univ. of Penna. Med. Bull., May, 1904, p. 90. See also Jour. Nerv. and Mental Dis., November, 1901, p. 595; Transactions Phila. Co. Med. Soc., 1904, p. 191; Transactions Coll. of Phys. of Phila., 1904; Transactions Amer. Neurol. Assoc., 1906, p. 31; Univ. of Penna. Med. Bull., July-August, 1905; Brain, 1906, p. 265.

location of these areas can be determined by referring to Figs. 78 to 81.

The Prefrontal Region.—That portion of the frontal lobes situated in front of the ascending and third frontal convolutions constitutes the *prefrontal region.* Here are thought to be located the *higher psychical centres,* those which have to do with the higher mental processes—viz., memory, attention,

FIG. 82



Side view of human brain, showing localization of functions. (Charles K. Mills.)

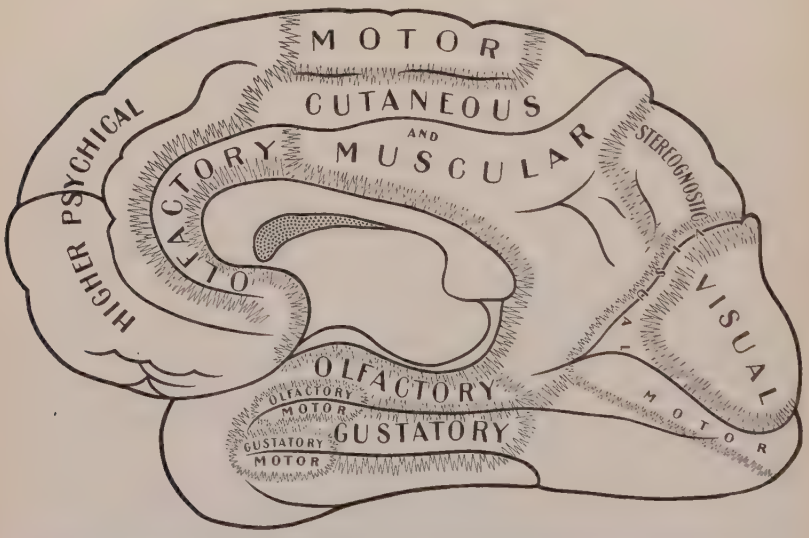
judgment, reasoning, and comparison. It has been thought by some[1] that these functions are limited to the left side in right-handed persons, and *vice versa.* According to Bianchi, it is that part of the brain where the various motor and sensory images deposited in the other centres of the cortex become coördinated and fused (an association centre). Lesions of the first and second frontal convolutions, especially

[1] Phelps, Amer. Jour. Med. Sci., April, 1902.

of the left side, may cause apraxia (p. 309). In addition, according to Munk, the trunk muscles have their cortical representation in this lobe.

Central Convolutions.—The centres for voluntary motor impulses are located in the entire length of the ascending frontal convolution, partly in the second frontal, movements of the head and eyes (Fig. 84) and in the paracentral lobule. It will be noticed that the centres for the leg movements are

FIG. 83



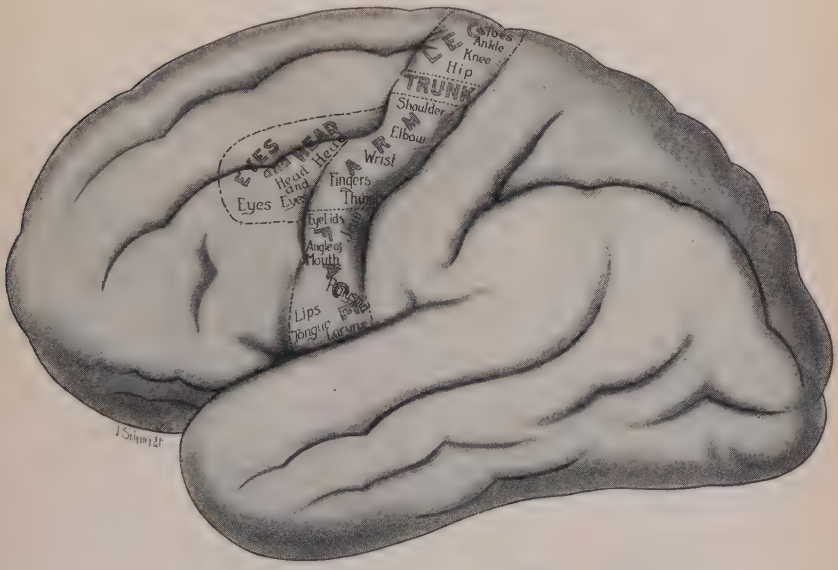
View of the mesial surface of the human brain, showing localization of functions. (Charles K. Mills.)

in the upper part of the convolution and in the paracentral lobule, that those for the arm are in the middle third, and that those for movements of the tongue, lips, and possibly the larynx and pharynx are in the lower part of the convolution.

Sensory impressions are probably received in the ascending parietal convolution, parietal lobe, and gyrus fornicatus. Impressions from the muscles and the stereognostic sense

(p. 50) are located in the superior parietal lobule (Figs. 82 and 83). Mills and Weisenburg[1] believe that the sensory region is divided into separated centres as the motor region is. The centres for olfactory and gustatory sensations are located in the uncinate region, the centres for hearing are in the first temporal convolutions (Figs. 82 and 83). Visual centres are described on p. 135 et seq., and those concerned in speech under aphasia (pp. 306 to 313).

FIG. 84



The subdivisions of the motor cortex. (Mills and Frazier.)

Destructive lesions involving the motor region cause a *paralysis*, which becomes spastic, of the muscles of the opposite side. It is not always complete, as the lesion may be limited to one or more centres only, causing, for instance, a monoplegia. Monoplegia is always due to a lesion of or near the cortex, when caused by a cerebral lesion.

[1] Transactions Amer. Neurol. Assoc., 1906, p. 31.

Also muscles which habitually act together, as the muscles of respiration, the facial muscles when used to express the emotions, the orbicularis palpebrarum, etc., are represented on both sides of the cortex, and a one-sided cerebral lesion does not cause their permanent paralysis, as the other side of the brain takes up the work.

Irritative lesions of the motor cortex causes localized spasms or spells of Jacksonian epilepsy (p. 439). Irritative lesions of the uncinate region causes sensations of taste and smell (hallucinations) frequently existing as auræ in peculiar epileptic attacks[1] (p. 437).

Occipital Lobe.—The cuneus and calcarine fissure are the primary visual centres (p. 135). Quadrant anopsia in which one-quarter of the field is obscured (p. 139) may be caused by a lesion here. This symptom is usually cortical. Hemiachromatopsia (p. 142) may also occur.

Centrum Ovale.—Lesions in this part may involve motor, sensory, or association fibers (Figs. 5 to 10). A lesion near the motor region of the cortex causes paralysis resembling in distribution that of a cortical palsy (subcortical lesion). Such lesions do not cause spasm; if they do occur, the spasm is due to the cortex becoming involved, and the paralysis precedes its development. In cortical lesions it is usually *vice versa*.

When the lesion is near the *internal capsule* the paralysis resembles that due to a lesion in that situation. There may also be hemianesthesia, hemianopsia, and, if on the left side, aphasia.

Corpus Callosum.—Symptoms indicative of a lesion in this situation are complete or partial hemiplegia, gradually extending to the opposite side, and later disturbances of speech, difficulty in deglutition, and dementia. Similar symptoms might, however, be due to other causes—viz., double cortical lesion. Apraxia (p. 309) has been caused by lesions here (p. 310).

Internal Capsule.—Through this pass within a small space the motor and sensory projection fibers, which pass to

[1] Brain, 1898, p. 580.

and from the cortex (Fig. 5). In the region of the "knee" of the capsule are the fibers which pass from the cortex to the nuclei of the motor nerves of the eye, and to the facial and hypoglossal nerves; also those which pass to the nuclei of the nerves which govern the other muscles concerned in speech. (See Anatomy, tenth nerve.) The pyramidal tract is next to these posteriorly, the fibers controlling arm movements being anterior to those for the leg. The fibers carrying impulses concerned in common sensation and those of special sense (vision, etc.) are posterior to these (Fig. 85). As the fibers of the pyramidal tract are here in a compact bundle, a lesion in this situation causes a hemiplegia upon the opposite side. Monoplegias are never caused by a lesion in this

FIG. 85

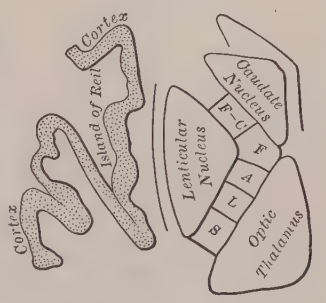


Diagram of horizontal section through the basal ganglia and internal capsule (left side), showing the position of the chief tracts in the internal capsule. The region of the capsule marked by the letters *L A F* is occupied by motor fibers: *L* corresponds to the leg fibers, *A* to the arm fibers, *F* to the face fibers (including fibers to face muscles and tongue). The region *F-C* contains the frontocerebellar tract (intellectual tract). The region marked *S* contains the general sensory tract from the opposite side and the fibers from the optic and olfactory nerves of the opposite side, sometimes called the "sensory crossway."

situation. A lesion in the hinder part of the posterior limb, causes hemianesthesia, which may involve the special senses, the defect of vision being a homonymous hemianopsia. Some motor paralysis would also likely be present.

Crus.—The third cranial nerve arises from the crus near the pyramidal tract. A lesion here would cause spastic hemiplegia upon the opposite side and paralysis of the muscles supplied by the third nerve upon the same side (Fig. 86). As the optic tract crosses the crus, it may also be involved, causing a homonymous hemianopsia.

If the *tegmentum* is involved without the motor tract, disturbances of common and muscular sensibility and ataxia of the opposite side occur, associated with paralysis of the

third nerve, and if near the aqueduct of Sylvius loss of associated upward movement of the eyes.

Optic Thalamus.—The thalamus is connected with the cortex of the frontal, parietal, occipital, and temporal regions. The optic tract and numbers of sensory fibers from the cord also end in it, a new neurone, the *thalamic cortico,* the axones of which reach the cortex, beginning here (p. 25). With the anterior corpora quadrigemina and lateral geniculate bodies the posterior part or pulvinar forms the primary centre for vision (p. 135). It probably also exercises an influence over various automatic functions as emotional control, facial expression, and bodily posture. Athetoid movements are probably due to disease of the thalamus (p. 37).

The *functions* of the *corpus striatum* are in dispute although lately the view has been advanced that lesions of the lenticular nucleus of the left side cause either anarthria (see Aphasia, p. 308) or dysarthria, and, therefore, contains centres which are concerned with movements that enter into speech. Paresis of either a monoplegic or hemiplegic type may also be caused by lesions here; it is not, however, so intense as that caused by either cortical or capsular lesions.[1]

Wilson[2] believes that pure lesions of this region do not cause paralysis, but that it "exercises a steadying effect on the action of the corticospinal system." Loss of this action causes hypertonicity and rigidity without the Babinski phenomenon and tremor on voluntary motion. The dysarthria occurring being due to hypertonicity of the muscles. Emotionalism may also be a symptom.

The Corpora Quadrigemina.—The *anterior tubercles*, together with the lateral geniculate bodies and pulvinar form what are known as the *primary visual centres* (p. 135). They are connected with the optic and motor oculi nerves, and control the reflex movements of the iris and ciliary muscles.

The *posterior tubercles*, together with the internal geniculate bodies, are connected with the *auditory nerves* and cerebellum,

[1] Mills and Spiller, Jour. Nerv. and Ment. Dis., September, 1907, p. 558, and October, 1907, p. 624.

[2] Brain, 1912, p. 295.

and control reflex movements connected with *hearing* and *sensations of space.*

The Cerebellum.—The *gray matter* of the cerebellum is situated in the cortex and in ganglionic masses in its substance; but excepting the *vermis* or median lobe, which appears to control most of its functions, the relations of this gray matter to the cerebellar functions are not well known. It is connected with the motor and sensory regions of both the brain and cord (see Motor and Sensory Tracts), the auditory nerve, some collections of nerve cells in the pons, and with the inferior olivary bodies of the medulla.

The **functions** of the cerebellum are still the subject of investigation. It is known to take an active part in the coördination of muscular movements and of maintaining our equilibrium and relation to surrounding objects. It also regulates automatic movements. According to Hughlings Jackson its disease causes paresis of the trunk muscles. It is also thought by many (Bastian and Hughlings Jackson) that it is cerebellar influence which maintains muscle tone.

Interference with its functions causes incoördination and inability to maintain the balance (cerebellar titubation), intense vertigo, often resembling that of Ménière's disease (p. 114), and weakness of the muscles of the back and neck. Each cerebellar hemisphere controls the muscles of its own side.

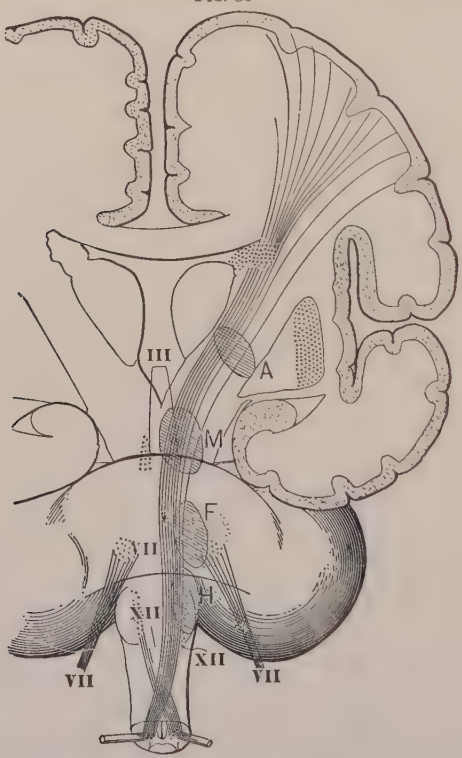
The Pons.—The gray matter of the pons consists of cells which form the nuclei or beginning of the peripheral neurones of the sixth, seventh, and motor division of the fifth cranial nerves. It also contains the ascending or sensory nucleus of the fifth nerve, and the motor and sensory tracts on their way from the cord to the cortex pass through it. There are other collections of nerve cells in the pons which are connected with the cerebellum and cerebral cortex.

If the lesion is in the *upper* part of the pons, where the fibers running to the cranial nerve nuclei have not yet decussated, the resulting paralysis would be similar to that due to cerebral disease.

Lesions situated below the middle of the pons usually involve the motor path, with one or more of the cranial

nerves mentioned above, causing a crossed paralysis, which, excepting when the third nerve is involved (see Crus), is always indicative of a lesion in the pons (Fig. 86). If situ-

FIG. 86



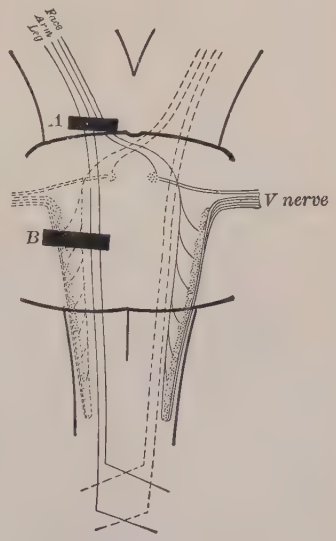
Showing the mechanism of alternate hemiplegias. A lesion at *A* causes complete hemiplegia by destroying the motor tract. One at *M* causes paralysis of the third cranial nerve (motor oculi) by destroying its nucleus or root on the same side, and paralysis of the arm and leg on opposite side. A lesion at *F* causes facial palsy on same side; hemiplegia on opposite side. In a lesion at *H* the hypoglossus would be affected on one side, with hemiplegia on the other. (Modified from Edinger.)

ated near the middle line, a lesion may cause paralysis of one motor tract and of both facial nerves. A destructive lesion involving either the nucleus of the sixth or posterior longi-

tudinal fasciculus causes conjugate deviation of the eyes to the side opposite and loss of associated lateral movements of the eyes to the same side as the lesion. The power of convergence will, however, be maintained (Fig. 39, *B*).

If the fillet and sensory root of the fifth nerve are involved, hemianesthesia results; if below the upper third of the pons, it is a crossed paralysis, the anesthesia of the face being on the same side as the lesion (Fig. 87). If the sensory root of the fifth escapes, the face is not involved. Bilateral disease may occur, causing various combinations of these symptoms.

Fig. 87



The sensory tract in the crus, pons, and medulla, showing nucleus and roots of *V* nerve. *A*, lesion causes right hemianesthesia; *B*, lesion causes alternating hemianesthesia; left face and right side of body.

Paralysis of the sixth and seventh nerves, without that of the auditory, indicates a lesion in the posterior part of the pons.

If the middle cerebellar peduncle becomes involved, forced movements occur. Acute lesions frequently produce hyperpyrexia and disturbances of respiratory and cardiac action.

Medulla Oblongata.—The nuclei, or cells of the second order neurones, of the cranial nerves, from the eighth to the twelfth inclusively, excepting that portion of the eleventh which comes from the cervical region of the cord, are situated in the medulla (Fig. 63). It is important to remember that the medulla and that portion of the pons containing cranial nerve nuclei are really portions of the cord, and that these cells are analogous to those cells found in the gray matter of the cord to be described, and that their functions are similar.

In the medulla, also, are situated the various reflex and automatic centres which control the circulatory and respiratory functions, visual movements, and secretion. The superior and inferior olivary bodies, masses of gray matter in the medulla, are connected with the cord, basal ganglia, and cerebellum. They have something to do with the functions of coördination and equilibrium.

Lesions high up may damage the pyramidal tract above its decussation and the twelfth nerve upon the same side as the lesion (Fig. 86).

Disease of the bulbar nuclei gives rise to the characteristic group of symptoms described under bulbar palsy. Acute lesions of the medulla usually cause death in a very short time.

Chronic lesions involving the floor of the fourth ventricle, as pressure from a cerebellar tumor, sometimes cause either polyuria or glycosuria, or both.

APHASIA

Definition.—By aphasia we mean the partial or complete loss of either the power of expression or of comprehension or both combined of any of the usual signs of language; not dependent upon lesions of the peripheral nerves or organs, but upon lesions of the cortical centres or tracts connecting them.

Etiology.—While most cases of aphasia are due to a destructive lesion on the left side of the brain in right-handed people, and *vice versa* — either tumor, acute hemorrhagic encephalitis, abscess, hemorrhage, or acute softening of the centres and tracts—it must be borne in mind that less commonly aphasia, usually transient, may be produced by *functional disturbances* — *i. e.*, fright, anger, epilepsy, migraine, arterial spasm (p. 323), neurasthenia, hysteria, toxemias of various sorts, as uremia, diabetes, gout, alcohol, mineral and vegetable poisons, and loss of blood. Some of these cases, especially the uremic, may be due to diseased bloodvessels produced by the action of the poison.

The Genesis of Speech.—Location of Centres.—For the proper understanding of aphasia some knowledge of the method of development of language is essential. The child learns to understand language before he can utter it. By the employment of the different senses, he perceives the different characteristics and appearance of an object. These percepts are stored away in the brain, and gradually the child learns to associate the name of the object which he hears applied to it with that object. The memory of the sound of this word is stored in a certain part of the brain (left first temporal convolution), so that hereafter when the word designating the object is used its various characteristics are at once brought into consciousness by a stimulation of the different centres where the memories of these characteristics are stored. Finally the child, probably by watching the muscular movements employed by others in pronouncing the word, learns to make them himself. These memories of the proper muscular movements necessary to pronounce a word are also stored away in a certain part of the brain.

The exciting of one of these centres excites the others; for instance, if we hear the sound of a bell, a mental image of the other characteristics of a bell is at once formed, and the proper word designating that image comes into consciousness. Then if it is wished to articulate the word, the properly coördinated muscular movements are made and the word is pronounced.

But even if the word is not pronounced, as in silent thinking, mental images of words enter into thought processes. Anyone can mentally recall the sound of a word, as he can also the visual appearance of it when written. This is being constantly done while thinking.

This *revival of words*, which in most cases is first in the auditory centre (first temporal convolution, is immediately followed by a revival of the *kinesthetic memory*, or the memory of sensory impressions resulting from the movements of the vocal organs during the utterance of words (glossokinesthetic centre or, as it is sometimes called, psychomotor centre, in the left third frontal convolution and insula). This is termed the *internal language*.

We learn to read by associating the visual appearance of certain symbols with certain sounds already familiar with the visual memories of these symbols. The so-called *visual memories* are stored in the cortex (angular gyrus). In reading aloud, the words which are primarily perceived in the visual centre at once call up the corresponding sound in the auditory centre from which the glossokinesthetic centre is stimulated and the memories of the required muscular movements are called up to articulate the word. These impressions are now sent to the centres for these muscles at the foot of the precentral convolution. When we read silently the internal language is employed in the same way.

In writing, which is usually next learned, the child learns to associate the visual perceptions of the letters with certain muscular movements of the fingers and arm required to make them. The memories of these movements are, as in the case of speech, stored up in a centre—the cheirokinesthetic centre—thought to be situated in the second frontal convolution on the left side. As in writing each letter is self-dictated by means of either spoken or internal language, the other centres involved in these processes are first excited. Reference to Fig. 88 will show the location of the centres mentioned and their probable connections.[1]

Varieties.—Based on whether the receptive or emissive function is affected, aphasia may be either *sensory* or *motor*.

Sensory aphasia (Wernicke's) is present when, the peripheral apparatus being intact, the patient is unable to under-

[1] Marie (La Semaine Médicale, May 23, 1906, p. 241) has denied that the third frontal convolution has anything to do with speech. He does not believe in the sensory images of language above described, but believes that the only speech centre is the zone of Wernicke, which consists of the supramarginal and angular gyri and the posterior part of the first two temporal convolutions. This region he believes is a purely intellectual centre and not one in which images are stored up. A lesion in this region causes the symptoms described above as sensory aphasia. What is generally known as motor aphasia as the above symptoms plus anarthria. Anarthria occurs when the lesion involves the "lenticular zone," which is an area comprised between a line passing in a transverse direction from the anterior fissure of the island of Reil to a corresponding point in the lateral ventricle and a line in a similar direction from the posterior fissure of the island of Reil to a corresponding point in the lateral ventricle. Within this area are situated the corpus striatum, external capsule, internal capsule, and cortex of the island of Reil. The anarthria or aphemia of Marie is characterized by loss of speech, with preservation of the understanding of words, reading, and writing.

stand the language which he has been accustomed to use (word-deafness or auditory aphasia); or is unable to recognize the meaning of the symbols, either written or printed, with which he was formerly familiar; in other words, is unable to read (word-blindness, alexia, or visual aphasia). The centre in the former is the left first temporal convolution and in the latter the left angular gyrus (Fig. 78).

Related to this is a form termed by Freund *optic aphasia*. In this the patient is unable to name an object with which he was once familiar, although he may be able to pronounce the name when he hears it. This symptom is termed *anomia*. Mills has placed what he terms the "naming centre" in the left second temporal convolution (*N* in Fig. 88), a lesion of which or of one of the tracts *SN*, *VN*, or *AN* would cause the symptom for the reason that the naming of an object depends upon the recognition of its various characteristics. A patient therefore may be able to name an object by seeing it when he could not by feeling it if the tract *SN* was diseased and *VN* not. A somewhat similar state is known as *agnosia*, by which is meant an inability to recognize familiar objects by any of the senses, although the power of perception is normal, thus a familiar sound is heard but it is not recognized, or a familiar object is seen and not recognized. One sense may be affected and not the others; thus we have auditory agnosia, tactile agnosia, etc. The latter is also known as *astereognosis* (p. 50); also related to aphasia is the symptom known as *apraxia*. Agnosia has been sometimes termed *sensory apraxia;* the term is usually applied to *motor apraxia* or *dyspraxia*. By this is meant the inability of an individual who has neither motor nor sensory paralysis, nor ataxia, to perform certain familiar purposive movements.[1] He knows what an object is, and what it is used for, can possibly name it, but has forgotten how to use it. An apparent motor apraxia may be based on agnosia, for instance, if a patient is unable to recognize an object when he sees it, it follows that he will not know what it is used for and consequently how to use it. The centre for motor apraxia has been placed

[1] Wilson, Brain, 1908, p. 164.

in the first and second frontal convolutions of the left side in right-handed people. The symptom has also been present in lesions of the corpus callosum.

Agnosia, apraxia and word-blindness are also known as *mind* or *psychic blindness.*

Motor aphasia (Broca's), or, as it is sometimes termed, *aphemia,* is present when the patient, the peripheral speech apparatus being intact, is unable, either completely or partially, to give expression to his thoughts and is due to a lesion in the left third frontal convolution or the fibers leading from it (Fig. 78).

Loss of the power of writing, not dependent upon weakness of the arm, is called *agraphia;* it is practically always, when present, associated with either aphemia or word-blindness. The centre has been placed in the second left frontal convolution.

Loss of the power of understanding or employing gesture or pantomime is termed *amimia.* Infrequently, either with or without defects of ordinary speech, there is loss of the power to produce or comprehend musical sounds; this defect is termed *amusia.*

In some cases the patient may be able to speak; but he skips words, uses wrong ones, and consequently talks confusedly. This is called *paraphasia* or *conduction aphasia.*

Each of these forms of aphasia is further divided into cortical and subcortical, according as the lesion is in the centre itself, or in one of the tracts leading either to or from it. The symptoms due to a cortical lesion differ somewhat from those due to a subcortical one. Thus we have cortical and subcortical motor aphasia, and similarly with the other forms.[1]

Cortical Auditory Aphasia.—In this form the lesion is at *A.* There would be loss of the power of understanding spoken words; nor could words be repeated or written from dictation. As stimulation of this centre is necessary to bring *GK* into

[1] While these arbitrary divisions have been made theoretically they practically do not maintain, as will be seen in studying the symptoms of the different forms. It will be noticed that so-called sensory and motor symptoms are more or less combined. Von Monakow (Neurol. Centralbl., November, 1906, p. 1026) explains this by the fact that when one centre is destroyed the functions of all tributary centres are more or less interfered with. He terms this "diaschisis."

activity, spoken speech would be defective, wrong words would be used, and paraphasia would result. For the same reason, the internal language being interfered with, the power of reading and writing would also be interfered with (tracts *V–A–GK* in Fig. 88).

FIG. 88

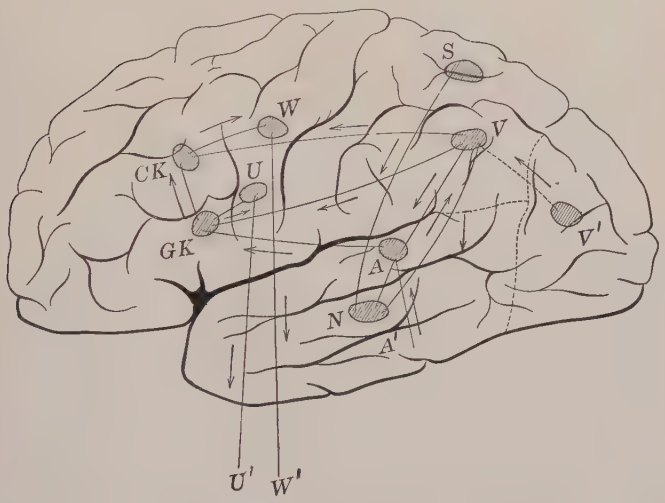


Diagram showing probable pathways of nervous impulses concerned in speech and writing:[1] *A*, centre for auditory word memories in first temporal convolution; *V*, centre for visual word memories in angular gyrus; *GK*, glossokinesthetic centre (Bastian), or psychomotor centre, at foot of third convolution; *U*, centres for muscles involved in articulation at foot of ascending frontal convolution; *CK*, probable centres for memory of muscular movements involved in writing, cheirokinesthetic centre of Bastian; *W*, centres for controlling muscles of arm and hand in ascending frontal convolution; *A–A'*, tract from cortex of temporal lobe (auditory centres) to centre for word memories; *V–V'*, tract from cuneus to centre for visual memories; *W–W'*, tract from arm and hand centres to cells in anterior horns of cord and peripheral nerves controlling these muscles (pyramidal tract); *U–U'*, tract from centres for muscles of articulation to centres in pons and medulla and nerves controlling those muscles (pyramidal tract). In speaking, impulses travel from *A–GK–U–U'*; in reading aloud, from *V–A–GK–U–U'*; in silent reading, from *V–A–GK*; in writing spontaneously, *V–A–GK–CK–W–W'*; in writing from dictation, *A–V–GK–CK–W–W'*; in copying, *V–CK–W–W'*; *N*, naming centre; *S*, stereognostic centre in parietal lobe.

[1] In right-handed persons these centres and tracts are situated in the left side of the brain; in left-handed persons the opposite is the case.

Subcortical Auditory Aphasia.—In this form the lesion is in the tract A–A'. The centre A is intact. The patient is unable to understand spoken words, or to repeat or write from dictation. There may also be some paraphasia, as the patient has not the benefit of hearing the word to know its correctness. The internal language being intact, word memories can be recalled (in centre A), and reading and writing are not interfered with (tract V–A–GK, Fig. 88).

Cortical Visual Aphasia.—The lesion is in the centre V; the patient is unable to read (alexia) aloud or silently; to write spontaneously, from dictation, or to copy. Volitional speech is not interfered with.

Subcortical Visual Aphasia.—Lesion in tract V'–V. Ability to read and to copy understandingly is lost. By means of tract V–CK he can copy mechanically. As the centre V is intact, visual memories can be recalled; hence the patient can write, but not with facility, he being in the position of one writing with his eyes closed.

Cortical Motor Aphasia.—The lesion is in the centre GK. Volitional speech and repeating words and reading out loud are lost. Owing to the interference with the internal language, silent reading and writing are also lost (V–A–GK, Fig. 88). Language is understood.

Subcortical Motor Aphasia.—This is the purest form of motor aphasia. Lesion in the tract GK–U. Volitional speech, repeating words, and reading aloud are lost. The internal language being intact, as the centre GK is intact, silent reading and writing are not interfered with. Language can be understood.

Motor and Sensory Aphasia.—It may happen that two or more of these centres are diseased, in which case there would be a combination of the symptoms above described. (See note, p. 310.)

Diagnosis.—In examining a case of aphasia we should examine:[1] (1) Voluntary speech; (2) exclamatory speech; (3) responsive speech; (4) associative speech, such as counting, saying alphabet, repeating familiar lines; (5) quality

[1] Dana, New York Med. Jour., August 10, 1907, p. 240.

of speech such as paraphasia, and confusion, stereotypy, jargon; (6) repeating spoken words; (7) indicating the number of syllables in a word—to show that the patient knows that it is a word; (8) writing; (9) writing to dictation; (10) copying; (11) singing (humming tunes); (12) gesturing; (13) understanding gestures; (14) understanding spoken words, sentences, and complex directions; (15) naming things seen, also felt, heard, touched, smelled, and tasted; knowing the proper uses of and manner of using objects; (16) naming abstract things and qualities; (17) recalling to mind objects named; (18) reading understandingly, aloud or silently; (19) reading letters or numerals; (20) reading aloud without understanding; (21) knowledge of the use of things; (22) general intelligence. It should also be ascertained that the patient is not deaf due to any anatomical lesion of the ear, and that there is no paralysis of the muscles of the lips, tongue, and larynx, and the eyes are normal.

Prognosis.—The prognosis is better in children than in adults, as in them education of the centres upon the other side of the brain takes place, and consequently restoration of the lost function. In adults more or less improvement often takes place. The outlook in sensory aphasia is better than in motor.

Treatment.—The treatment is in the first place that of the lesion causing it; in the second place, of the symptoms. This consists of reëducation by the usual methods employed in teaching children to speak, read, and write.

AFFECTIONS OF THE BLOODVESSELS

The Blood Supply of the Brain.—The **external carotids** furnish the blood to the scalp, skull, and dura mater.

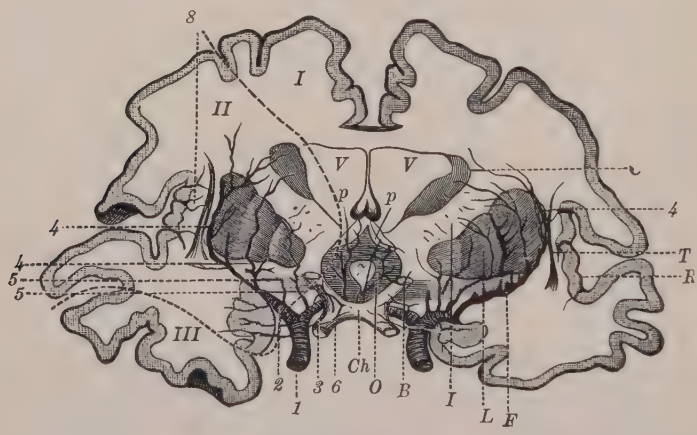
To the neurologist the **middle meningeal,** which arises from the internal maxillary, is the most important branch. It supplies that part of the dura situated over the sensorimotor region, and is frequently the source of hemorrhage due to traumatism (p. 318).

The **internal carotids** and **vertebrals** furnish the blood to the pia mater and brain substance.

Each *internal carotid* divides into two branches: the anterior and middle cerebrals. The two former are united by the anterior communicating.

The *vertebrals* unite to form the basilar, which divides into the two posterior cerebrals; these are united with the internal carotids by the posterior communicating arteries. This arrangement is known as the *circle of Willis* (Plate III).

FIG. 89



Transverse section of the cerebral hemispheres, about 1 cm. behind the optic commissure: *Ch*, chiasma; *B*, section of the optic tract; *L*, lenticular nucleus; *I*, internal capsule; *C*, caudate nucleus; *E*, external capsule; *T*, claustrum; *R*, island of Reil; *V*, *V*, section of the lateral ventricle; *P*. *P*, anterior pillars of the fornix; *O*, gray substance of the third ventricle. *Vascular areas:* 1, internal carotid artery; 2, Sylvian artery; 3, anterior cerebral artery; 4, 4, external arteries of the corpus striatum (lenticulostriate artery); 5, 5, internal arteries of the striatum (lenticular arteries); 6, arteries of the chiasma; 7, arteries of the commissure and anterior pillars of the fornix; 8, arteries of the convolutions of the insula and claustrum. The optostriate artery is not represented in the figure. (Duret.)

Central Arteries.—From the circle of Willis and the beginnings of the anterior, middle, and posterior cerebral arteries are given off groups of vessels, known as the *central arteries*. These supply the basal ganglia and adjacent white matter. They are all *end arteries* consequently do not anastomose, and are the vessels usually affected in cerebral hemorrhage in adults (Fig. 89).

The researches of Beevor[1] have conclusively settled the arterial supply of different parts of the brain and changed some former views. The supply of the more important regions is as follows:

Internal capsule, anterior one-third of posterior limb is supplied by the posterior communicating; the posterior two-thirds of the posterior limb by the anterior choroid. These are both branches of the internal carotid (Plate III). It also receives branches of the middle cerebral.

Corpus striatum, principally by branches of the middle cerebral.

Optic thalamus, principally by branches of the posterior cerebral.

Corpora quadrigemina, by the posterior cerebral.

Crus cerebri, by posterior cerebral and anterior choroid.

Cortical Arteries.—The anterior, middle, and posterior cerebrals themselves are distributed over the cortex in the pia mater; from there branches penetrate into the cortex and adjacent white matter. These are known as *cortical arteries.* Each of the cerebrals has a somewhat distinct area of the cortex which it supplies (Figs. 90 to 93). The anterior cerebral supplies the greater part of the frontal lobes and the mesial surface back to nearly the parieto-occipital fissure (Figs. 90 to 93). The middle cerebrals, the central convolutions except the upper parts which are supplied by the anterior (Figs. 90 to 93) (sensorimotor region) and island of Reil and some of the temporal and parietal lobes; the posterior cerebral supplies the occipital and part of the temporal lobes (Figs. 90 to 93). There is some anastomosis between the branches of these vessels; so that if, for instance, a branch of the anterior cerebral were plugged, some vicarious circulation would occur from the middle cerebral.

The pons and cerebellum are principally supplied by branches from the basilar; the medulla, and part of the cerebellum by branches given off from the vertebrals before they unite to form the basilar, principally by the posterior

[1] Brain, 1907, p. 403, and Phil. Trans. Royal Soc. of London, 1908, vol. cc.

FIG. 90

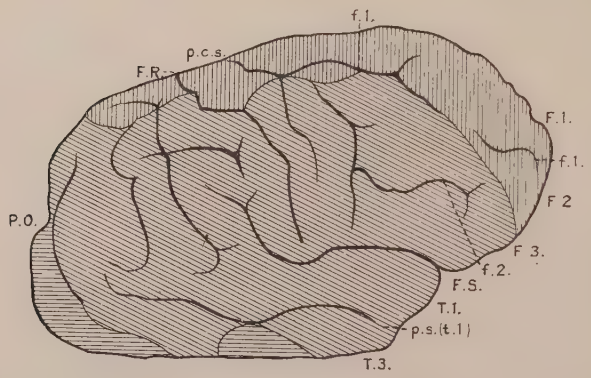


FIG. 91

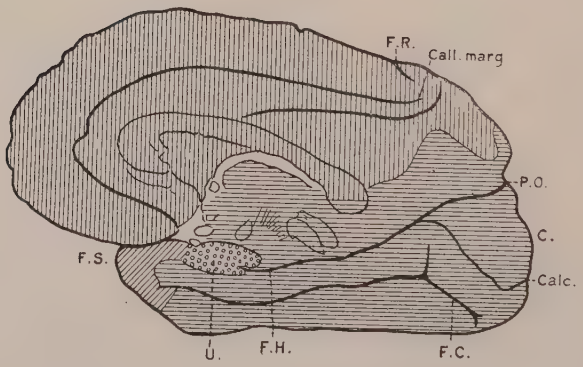


FIG. 92

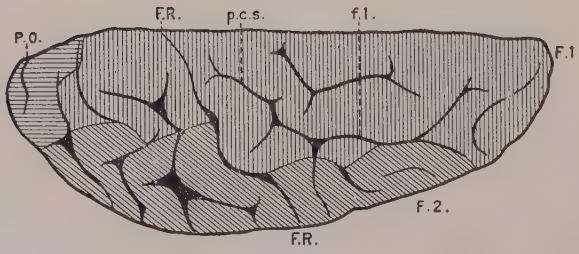
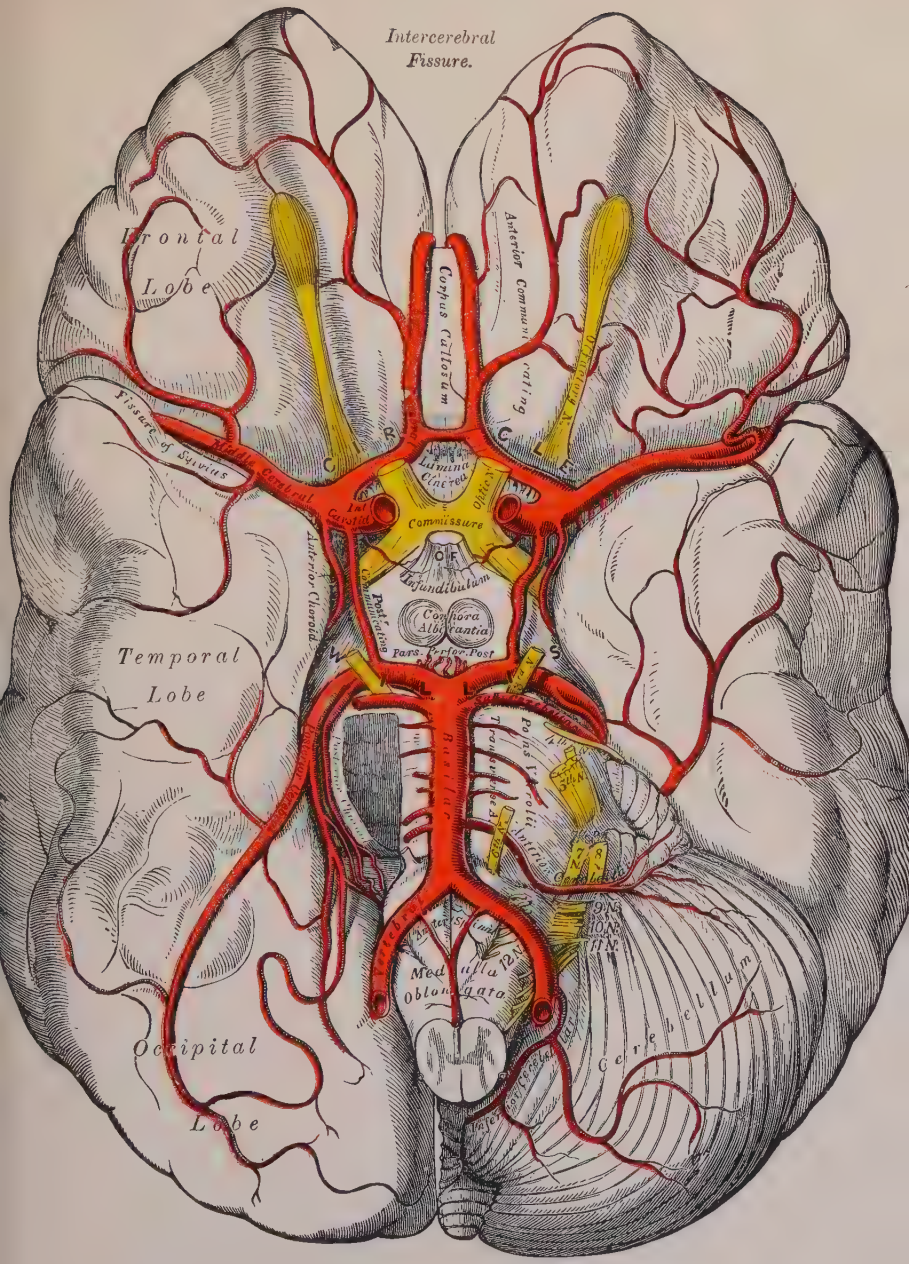


PLATE III

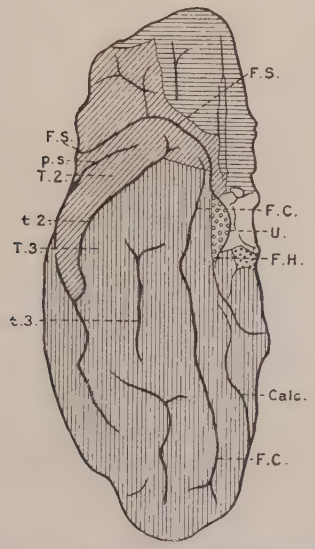


The Arteries of the Base of the Brain.

The right half of the cerebellum and pons have been removed. It will be noticed that the two anterior cerebral arteries have been drawn at a considerable distance from each other; this makes the anterior communicating artery appear very much longer than it really is.

inferior cerebellar artery (Plate III). Interference with its circulation causes ataxia and bulbar symptoms (p. 334).

FIG. 93



Figs. 90 to 93 are the external, median, superior, and inferior surfaces respectively of the right human hemisphere. The area of the anterior cerebral artery is represented by lines drawn coronally across the long axis of the hemisphere. The area of the middle cerebral artery is represented by lines drawn diagonally. The area of the posterior cerebral artery is represented by lines drawn parallel to the long axis of the hemisphere. The area of the anterior choroid artery is represented by small rings, and that of the posterior communicating by dotted shading. (After Beevor, in Brain.)

Explanation of Figures. *C.*, cuneus; *Calc.*, fissura calcarina; *Call. marg.*, callosomarginal fissure; *F.*1, gyrus frontalis superior; *F.*2, gyrus frontalis medius; *F.*3, gyrus frontalis inferior; *f.*1, superior frontal sulcus; *f.*2, inferior frontal sulcus; *F.C.*, fissura collateralis; *F.H.*, fissure hippocampi; *F.R.*, fissura Rolandi; *F.S.*, fissura Sylvii; *p.c.s.*, precentral sulcus; *PO.*, fissura parieto-occipitalis; *p.s.*, parallel or first temporal sulcus; *T.*1, gyrus temporalis superior; *T.*2, gyrus temporalis medius; *T.*3, gyrus temporalis inferior; *t.*2, second temporal sulcus; *t.*3, third temporal sulcus; *U.*, uncus.

The Veins.—The blood from the convexity and median surface of the cerebral hemispheres is collected by veins which empty into the longitudinal sinus. That from the inferior and lower surfaces reaches the cavernous, superior

petrosal, and lateral sinuses. The venous blood from the distribution of the central arteries enters the veins of Galen and from thence the straight sinus. The blood from the cerebellum partly reaches the straight and partly the superior petrosal and lateral sinuses, while that from the under surface of the cerebellum and the pons and medulla enters the inferior petrosal, lateral, and occipital sinuses.

MENINGEAL HEMORRHAGE

Hemorrhages into the meninges of the brain may occur in various situations—viz., into the meshes of the pia mater, which would, of course, include the brain cortex; into the subdural space, and between the external layer of the dura and the cranium. It may be *spontaneous,* but is usually *traumatic.*

Spontaneous Meningeal Hemorrhage.—Etiology.—Spontaneous hemorrhage when it occurs in adults, which it rarely does, is due to the same causes that produce intracerebral hemorrhage (p. 322). That occurring in children is more common, and has been discussed on page 249.

Symptoms.—The symptoms depend on the situation and size of the hemorrhage. If subdural, they may consist of sudden severe headache, vomiting, and signs of meningeal irritation. (See Meningitis.) These symptoms occurring in a plethoric person after eating, or muscular exertion, are suspicious.

If *pial,* the symptoms are headache, convulsions, possibly of Jacksonian type, followed by coma, and paralysis situated according to the location of the hemorrhage.

The **treatment** is similar to that employed for cerebral hemorrhage (p. 331).

Traumatic Meningeal Hemorrhage.—Etiology.—This is due to the rupture of a vessel, most frequently a branch of the middle meningeal artery, caused by a blow upon the head, which in most cases also causes fracture of the skull.

Symptoms.—When the hemorrhage is from the middle meningeal the clot is extradural, and the usual history is that

for a varying length of time—a few minutes to several hours[1] —after receiving the blow the patient, with the exception of more or less evidences of concussion, presents very few or no symptoms. Then he gradually becomes stupid, and finally comatose. Motor disturbances then make their appearance. More or less marked hemiplegia, which may or may not be preceded by twitchings of the leg or arm, or both, is the most common; or there may be muscular twitchings and rigidity of both legs or of legs and arms. Paraplegia has been present in a few cases. The paralysis may be very slight and soon disappear, or there may be no motor symptoms discoverable. Other localizing symptoms, as sensory disturbances, aphasia, etc., may be present. The pupil upon the injured side is frequently dilated (Hutchinson's pupil), and when present indicates that the hemorrhage has reached the base and is pressing upon the third nerve. Cushing[2] has called attention to value of studying the fundus of the eye in suspected cases of this nature. Venous stasis and beginning edema of the disk can be recognized upon the side of the hemorrhage (p. 137). The pulse at first is slow, becoming rapid later. Cushing[3] has also called attention to the elevation of blood pressure as a symptom of compression. This is nature's method of increasing the supply of blood to the vital centres in the medulla which otherwise become anemic due to its compression against the side of the foramen magnum by the increased intracranial pressure. A rapid fall of blood pressure in these cases is therefore a bad symptom. The respiration is slow, labored, possibly stertorous, and the temperature may be elevated.

In severe cases concussion symptoms complicate the case, and there may be no interval of retained consciousness.

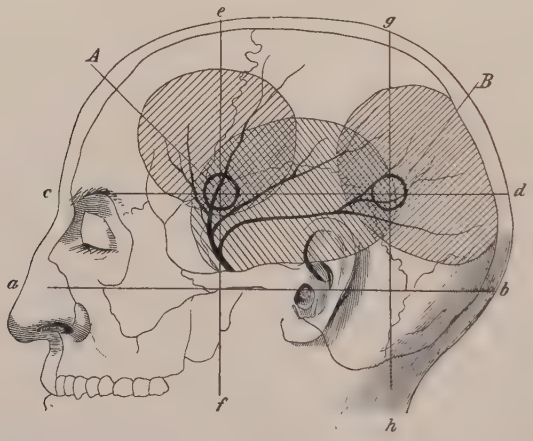
When the hemorrhage is *subdural,* it may occur from a pial vein or sinus, and the symptoms (coma and paralysis) usually but not always develop quicker,[4] and are apt to be more marked. Blood will likely be found in the fluid

[1] Cases have been reported in which days elapsed before symptoms appeared.
[2] New York Med. Jour., January 19 and 26, 1907; February 2, 1907.
[3] Amer. Jour. Med. Sci., June, 1903, p. 1017.
[4] The writer has had a case of this nature in which the interval was two days.

obtained by lumbar puncture. There is also more shock, the temperature after the injury going frequently to 95° or 96°, followed by a rise.

Diagnosis.—The diagnosis is based upon the symptoms above detailed, following a blow upon the head. While evidence of the trauma is usually visible upon the scalp, it may be absent.

FIG. 94



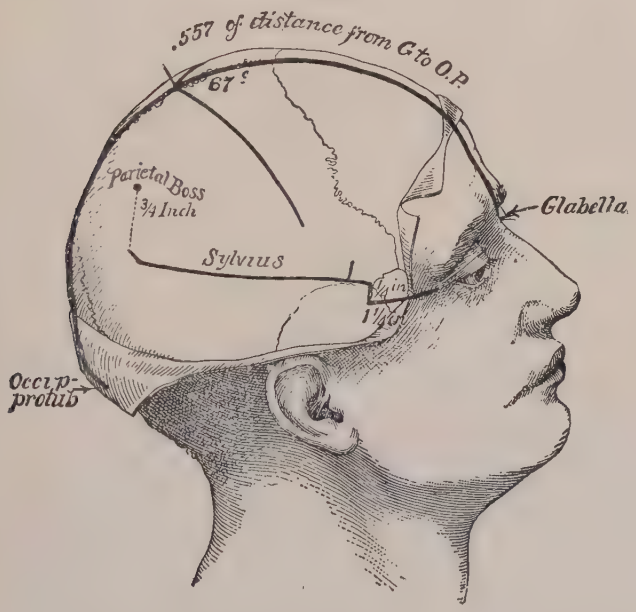
Points at which to trephine in hemorrhage from the middle meningeal artery: *a b*, horizontal line drawn through the meatus; *c d*, through the eyebrow; *e f*, vertical line, ¾ cm. behind the external angular process; *g h*, immediately behind the mastoid process. The intersections *A* and *B* mark the points for trephining to find respectively the anterior and posterior branches of the artery. (After Kronlein, Esmarch, and Kowalsky.)

Walton[1] has called attention to the fact that a severe concussion, attended by contusion and laceration of the brain cortex and subarachnoid serous exudation at the seat of injury, may, if the injury is in the motor region, produce symptoms simulating meningeal hemorrhage. As aids in distinguishing this condition from hemorrhage, he gives the following: (*a*) An atypical course; (*b*) absence of steadily increasing coma; (*c*) appearance of sensitiveness to

[1] Amer. Jour. Med. Sci., September, 1898, p. 267.

pain on manipulation of the head, even after the unconsciousness is so great that questions are not answered; and (*d*) the occurrence of the symptoms in children and young adults and in alcoholics. Such cases often recover without operation. But if the symptoms either do not improve

FIG. 95



The relation of the fissures of Rolando and Sylvius to the skull.[1] (Starr.)

very soon or increase, operation is safer than waiting. A fall of blood pressure without improvement otherwise demands immediate decompression.

It should also be remembered that sometimes the hemorrhage may take place from the side opposite the one that received the blow.

[1] 0.557 of the distance between the glabella and the occipital protuberance may be obtained for practical purposes by taking a point one-half inch posterior to half the distance between these points. The fissure of Rolando is about three inches long.

Prognosis.—The prognosis depends upon the seat of the hemorrhage, *i. e.*, supradural or subdural, and whether its seat can be reached by surgical means.

In *supradural hemorrhage*, if recognized early, the prognosis is hopeful. *Subdural hemorrhages* are harder to recognize, and when they are the prognosis is not so good. The possibility of extensive laceration of and hemorrhage into the brain substance coexisting must be borne in mind.

Treatment.—The treatment is surgical, and consists of making an osteoplastic flap over the probable seat of hemorrhage, removing the clot, and securing the vessel. In deciding the location we are guided by the symptoms: if motor symptoms are present, the opening is first made upon the opposite side. If no clot is there found the operation should be repeated upon the other side. All cases in which there is a possibility of such hemorrhage having occurred should at once be operated upon. In making the flap it should usually be located to expose the fissure of Rolando (Fig. 95), but may be moved more or less anterior or posterior if the symptoms warrant it.

If no localizing symptoms are present, operate at the seat of injury. For the *method* of locating the artery see Fig. 94.

APOPLEXY

The symptoms produced by either cerebral hemorrhage, embolism, or thrombosis are familiarly termed *apoplexy*.

Cerebral Hemorrhage.—Etiology.—At the time of birth and during infancy there is some tendency to cerebral hemorrhage (p. 249). From this period until the age of forty the liability is small; after this it progressively increases. More cases occur in cold weather and at high altitudes. Hereditary influence, insomuch as it predisposes to renal and arterial disease, is a factor. The most important predisposing cause is chronic alcoholism; next syphilis, then gout; chronic lead poisoning is also responsible for some cases. Because they produce fatty changes in the vessel walls, hemorrhage sometimes follows the infectious fevers. It may also, rarely, com-

plicate purpura hæmorrhagica and scurvy. Violent exertion, as the lifting of heavy weights, straining at stool, the act of coitus, and mental excitement, may act as exciting causes. Overindulgence in alcohol also may act in this way.

Transient apoplectiform attacks due to sudden congestion occur in multiple sclerosis, brain tumor, and paretic dementia. They also occur in those suffering from arteriosclerosis due to lack of blood owing to the narrowed lumen of the vessels (which may be due to spasm), and in uremia.

Morbid Anatomy and Pathology.—The hemorrhage (though possibly trumatic in rare cases) is almost always due to disease of the bloodvessels. This may consist of:

1. A *degenerative arteritis*, which results in the formation of miliary aneurysms, the rupture of which causes the hemorrhage. The degeneration first attacks the media. The aneurysms occur most frequently upon the branches of the middle cerebral (p. 315). On section of the brain substance they appear as localized, dark bodies about the size of a pin's head.

2. *Endarteritis* and *periarteritis* may lead to apoplexy by predisposing to the formation of either miliary aneurysms or coarse ones of the larger vessels of the circle of Willis.

3. *Fatty degeneration* of the small vessels occurring in purpura, scurvy, leukocythemia, marasmic conditions, and acute infectious diseases. Atheroma is usually found in the larger vessels.

4. Either rupture or thrombosis may occur some little time after an injury to the head (delayed or late apoplexy).[1] This may be due to an injury to the vessel, and a secondary rise of blood pressure causes the hemorrhage or the concussion may cause injury to the vessels which undergo endothelial degeneration and thrombotic processes are set up. Traumatism may also cause a latent arteriosclerosis to become active.

The parts affected in hemorrhage, in the order of frequency, are: the caudate and lenticular nuclei, meninges and cortex, centrum ovale, optic thalamus, pons, cerebellum,

[1] Allen, Jour. Nerv. and Ment. Dis., December, 1908, p. 763.

and medulla. Ventricular hemorrhages are usually secondary to hemorrhage into the neighborhood of the basal ganglia.

After a hemorrhage there is first coagulation of the blood, which soon begins to soften and be absorbed. The inflammation occurring about the clot usually causes the formation of a fibrinous wall about it, which forms a cyst with fluid contents. In other cases, instead of the formation of a cyst, there is proliferation of connective tissue and the formation of a pigmented scar. The acute symptoms are due, first, to destruction of the parts by the hemorrhage, and, second, to pressure upon and irritation of the neighboring parts.

Secondary degenerations follow, due to the cutting off of nerve fibers from their parent cells, which in most cases, as hemorrhage in the region of the internal capsule, is most common, would involve the pyramidal tract and be the cause of late rigidity and increased reflexes (the inhibitory influence of the cortical cells being cut off). (See Fig. 96.)

Symptoms.—The symptoms are divided into those of the attack and acute stage following, and, if the patient survives, the succeeding chronic state. The following description applies to the usual form of attack—that due to hemorrhage from one of the lenticulostriate arteries (Fig. 89).

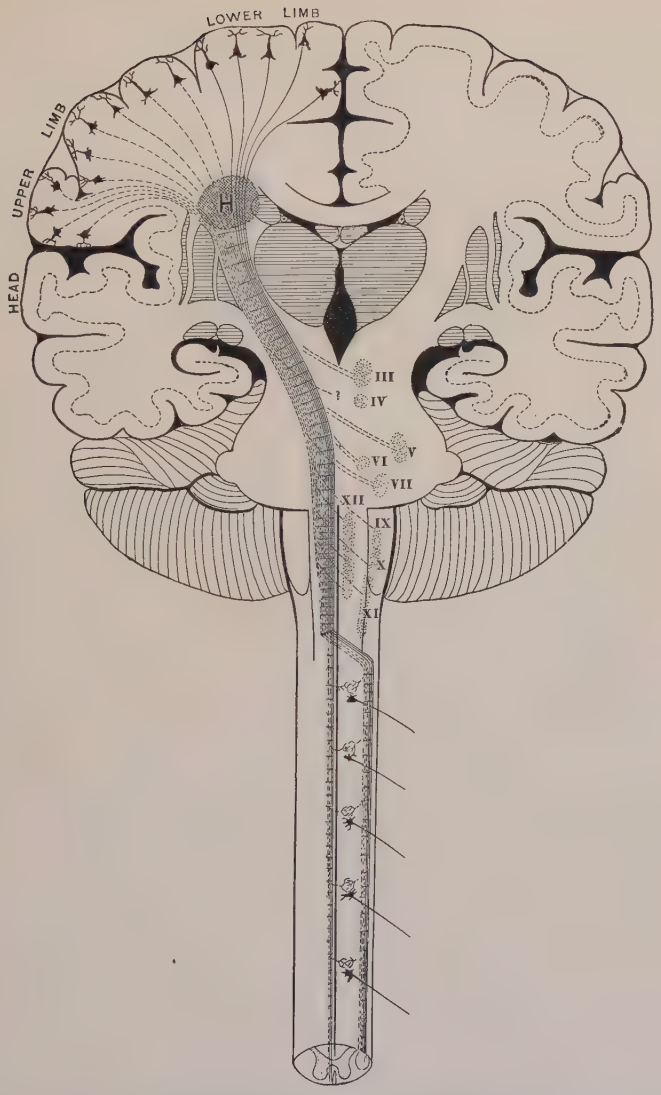
Prodromal symptoms are rare, excepting in cases due to syphilis: patients may complain for a few days previous of vertigo, numbness of the hand or foot, mental confusion, loss of memory, and headache.

The attack is always sudden, and may be accompanied by either convulsions and coma, coma alone, or consciousness may be preserved.

Convulsions are rare in cerebral hemorrhage, and when present usually indicate hemorrhage from a cortical artery (middle cerebral), as the cortical cells would be then irritated.

The usual mode of onset is for the patient, without warning, to become suddenly dizzy, lose consciousness, and fall. The face will be flushed, the pulse slow, full, and tense; the blood pressure increased, the breathing stertorous; the eyeballs fixed or deviated to the paralyzed side (conjugate

FIG. 96



Showing the secondary degeneration which occurs with cerebral hemorrhage or softening, and which follows the course of the motor tracts into the spinal cord. *H*, site of lesion. The continuous lines are fibers going to the legs, the dotted are those going to arms and motor cranial nerves. (Modified from Van Gehuchten.)

deviation, page 146); the pupils contracted and irresponsive to light, the skin bathed in sweat and the limbs relaxed, those of the side which is paralyzed being more so than those of the other; the mouth will also probably be drawn toward the unparalyzed side. The urine may be retained, or, with the feces, evacuated involuntarily. If the hemorrhage is severe, the temperature may be subnormal during the first few hours. After this, as a rule, the temperature rises, that of the paralyzed side being frequently 1° to 2° higher than the other. This condition is maintained throughout the acute stage.

In some cases the coma continues, the respiration assumes the Cheyne-Stokes type, the temperature rises, swallowing is impossible, and death occurs in a few days. In other cases the patient may partially regain consciousness, but is stuporous, with perhaps mild delirium. He is restless and complains of headache. This may continue for two or three weeks, when the temperature rises above normal, bedsores develop, pneumonia sets in, consciousness is again lost, and death ensues.

In the favorable cases the coma passes away in a few hours, although the patient may be mentally confused; on one side will be found paralysis of the arm, leg, and lower part of the face, the tongue when protruded pointing toward the paralyzed side. The speech is thick and indistinct; in hemorrhage upon the left side there may be aphasia. Slight anesthesia may also be found. In milder cases the patient may not lose consciousness, merely being confused for a time.

At first the paralysis is flaccid[1] and the deep reflexes lessened or absent, but in a few days they became increased and the Babinski reflex is present. For the first few days the temperature is above normal, being higher in the paralyzed side, and then falls to normal. If the temperature continues to rise after the fourth or fifth day it is a sign of extension of the hemorrhage or severe inflammatory reaction.

[1] In some cases rigidity of the paralyzed muscles appears at once, and is then known as "early rigidity." It is due to irritation and soon passes off. It must not be confounded with that appearing later, which is due to secondary degeneration (p. 324).

The Chronic Stage.—At the end of three or four weeks, if symptoms of cerebral irritation have disappeared, the chronic stage begins. By this time the paralysis of the face and tongue has either improved or disappeared, and some power of movement of the arm and leg has returned. The improvement is usually noticed first in the leg, which recovers more rapidly and to a greater extent than does the arm. The muscles near the trunk also regain more power than do the distal ones. Sensory symptoms have disappeared. The muscles that act bilaterally are most slightly involved. About this time, or possibly earlier, rigidity of the muscles develops (this is termed late rigidity), which usually increases until contractures affect the paralyzed limbs, producing deformities of greater or less degree (Fig. 60). The deep reflexes are exaggerated. In walking the toe is dragged and the leg swung from the hip in the arc of a circle. Rarely there may be a flaccid paralysis with absent reflexes (p. 36). The muscles do not waste[1] to any great extent; some atrophy may, however, be apparent. In the hemiplegia of adults the disorders of movement, athetosis, etc. (which frequently occur in children and are described on pp. 37 and 251), but rarely occur. There may, however, be intermittent tonic spasms affecting different groups of muscles to which the term "Hemihypertonia Postapoplectica" has been applied[2] (p. 37).

Unless the posterior part of the capsule is involved, hemianesthesia is not present. In some cases pain, either spontaneous or on pressure, in the arm and leg is complained of. In the majority of instances where this has been a symptom a lesion has been found in the optic thalamus.[3] Vasomotor disturbances, as cyanosis, sweating, etc., may be present. The mental condition is usually more or less affected, the patient being emotional and irritable; the memory fails, and in some cases complete dementia develops.

[1] This is the fact in nearly all cases. A few, however, have been reported in which there was atrophy without demonstrable lesion of the peripheral neurone.

[2] Spiller, Philadelphia Med. Jour., December 16, 1899.

[3] Rhein, Jour. Nerv. and Ment. Dis., October, 1912, p. 660.

Ventricular hemorrhage may be primary, but usually is secondary to hemorrhage into the cerebral substance. In the former case the symptoms are very severe, and as the blood soon fills all the ventricles, the centres in the pons and medulla are interfered with and death soon occurs. In the latter the original symptoms may be either improving or else in *statu quo* when a marked exacerbation occurs, the temperature falling, the pulse becoming slow, and the respiration labored. Conjugate deviation returns, often in the opposite direction to that in which it had been. Rigidity becomes marked, often both sides being affected, and there may be convulsions. In a few hours the temperature rises, the face becomes cyanosed, respiration more difficult, the chest fills up, and death soon occurs from paralysis of respiration.

Ingravescent apoplexy is a special form in which the patient is seized with sudden vertigo, headache, and vomiting, but without loss of consciousness. Hemiplegia, with hemianesthesia, sets in and in about twenty-four hours the patient becomes stupid and finally comatose, with death in from three to five days. The vessel ruptured is one of the branches of the external lenticular artery, the hemorrhage being mainly at first in the external capsule, breaking through the white matter, and finally reaching the lateral ventricle.

Hemorrhage into the crus may cause the characteristic symptoms of paralysis of the side opposite the lesion, and of the third nerve on the same side (Fig. 86). Hemianesthesia may also be present from extension toward the tegmentum and internal capsule. It may also extend into the upper part of the pons, causing the characteristic symptoms of a lesion there.

Hemorrhage into the Pons.—This is accompanied by loss of consciousness, frequently with spasmodic movements of the limbs, marked contraction of the pupils, slow respiration, high temperature—103° to 104°. The facial and ocular muscles and those of articulation and swallowing may be involved, and there is more or less marked paralysis of motion and sensation, usually unilateral. Crossed paralyses frequently occur (Fig. 86), and conjugate deviation in the

early stages is away from the paralyzed side (irritation) (Fig. 39 *B*).

Hemorrhage in the Cerebellum.—These are difficult to recognize. There may be a preliminary period of headache; or the patient may fall in a state of coma, as in hemorrhage elsewhere. Vomiting frequently occurs and is persistent. If hemiplegia occurs, it is on the side of the lesion if pressure is made on the medulla, if on the pons it is on the opposite side. It usually is absent. Palsies of cranial nerves arising from the medulla and pons may also occur from pressure. Respiration and swallowing are frequently much affected, as the fourth ventricle is almost sure to be involved. Death usually occurs.

Diagnosis.—Cerebral hemorrhage, when its onset is marked by **coma**, must be distinguished from coma due to either concussion or compression of the brain, acute alcoholism, uremia, diabetes, comatose state in pernicious malaria, opium poisoning, epilepsy, and hysteria; and when none of these are found to cause the symptoms, from acute softening of the brain due to blocking of a cerebral vessel by embolism or thrombosis. Both concussion and compression of the brain would be preceded by traumatism, evidence of which should be sought for. In these cases the possibility of meningeal hemorrhage (p. 318) must not be lost sight of.

Alcoholic coma is characterized by the odor of alcohol upon the breath, although this may be present, of course, in a case of hemorrhage; incompleteness of the coma; absence of localized paralysis, of the Babinski reflex, of unequal and contracted pupils, of conjugate deviation, and of low or unequal temperature.

Uremic coma would be characterized by the presence of albumin and casts in the urine, and a possible low specific gravity, urinous odor of the breath; facies common to chronic nephritis, *i. e.*, pallor, edema, albuminuric retinitis, etc.; absence of unequal pupils, of localized paralysis, conjugate deviation of the eyes, and differences of temperature upon the two sides. In addition to coma, uremic poisoning may produce a hemiplegia, the development of which may or may not be preceded by convulsions (see also p. 323); or

hemiplegia may develop without previous coma. Aphasia may also be so caused. These cases, while functional in origin, due to the action of the poison upon the cells, and no cerebral lesions being found after death, may closely resemble true apoplexy. They are usually transient, and such attacks associated with other evidences of nephritis would be suggestive. The occurrence of convulsions would be in favor of uremia. Of course, it must not be forgotten, that diseased kidneys may be present in cases of true apoplexy.

Diabetic coma is distinguished by the presence of sugar in the urine, the odor of acetone on the breath, and the absence of paralysis.

The comatose state in *pernicious malaria* could be determined by an examination of the blood and the existence of high fever following a chill.

Cerebellar hemorrhage is especially liable to be confounded with diabetic coma, as glycosuria frequently occurs in cerebellar lesions. Evidences of paralysis would be in favor of hemorrhage. A history of the other symptoms characteristic of diabetes could be ascertained if the attack was one of diabetic coma.

Opium poisoning is accompanied by marked and equal contraction of the pupils, extremely slow respiration, and absence of localized paralysis and of elevated temperature.

Pontine hemorrhage is especially apt to be confounded with opium poisoning; the high temperature and convulsions, if present, occurring in it are valuable diagnostic points.

In coma following epileptic convulsions there would be a comparatively rapid return of consciousness, absence of paralysis, and the history obtained, if one can be, of previous attacks.

Hysterical coma and **hemiplegia** will be described under Hysteria (pp. 459 and 472).

In cases seen **without previous history,** the diagnosis may sometimes be most difficult. The possibility of any of the above causes operating must be borne in mind and eliminated if possible. More or less transient apoplectiform seizures occur in those suffering from *arteriosclerosis, multiple sclerosis, brain tumor, general paresis,* and some other forms

of mental disease. A *syphilitic meningitis* may also simulate apoplexy, but such cases are usually preceded by headache, and the coma in most instances develops gradually; evidences elsewhere of syphilitic infection should be sought for. In many cases the diagnosis will have to be made by excluding the above-mentioned causes of coma. The diagnosis from *acute softening* is discussed on page 335.

Prognosis.—The first attack is in most cases recovered from; but after one attack there is always liability to another. The prognosis becomes worse in each succeeding attack.

Symptoms of bad omen are: profound and long-continued coma; coma continuing four days; an initial subnormal or a continued elevated temperature; the development of delirium; the occurrence of the attack in a chronic alcoholic or subject of renal disease; development of bed-sores; and continuance of loss of control over bowels and bladder. Cerebellar and pons hemorrhages and those which break into the ventricles are nearly always fatal. As regards return of function, the prognosis is doubtful; in some cases very fair power may return with, however, more or less rigidity, others remain practically helpless; this is especially so in those cases in which the limbs remain flaccid and the reflexes absent. The arm usually remains the most affected.

Treatment.—In treating the attack the patient should be recumbent, with the head moderately high, and it is well for a considerable part of the time for him to lie on his side so that the paralyzed tongue can fall forward. The room should be quiet and darkened. Ice may be applied to the head and cloths wrung out in mustard (cupful to a pail of water) and hot water wrapped about the legs. In a robust subject, with evidences of great excitement of the circulation, the face red or cyanotic, when it is certain that a high blood pressure preceded the stroke, venesection is of advantage.

It is well to remember that a moderate increase of blood pressure may be beneficial (p. 319) and that the medicinal measures formerly recommended (veratrum viride, nitroglycerin, purging with croton oil) are of doubtful value. These measures are, however, still recommended by authori-

ties. If necessary the patient must be catheterized. After the first few hours and until the commencement of the chronic stage, the treatment consists in keeping the mouth and nasal passages clean with antiseptic solutions, seeing that the position is changed at intervals, and meeting such symptoms as may arise. It is unnecessary to give food during this time. The patient should remain in bed for from two to four weeks according to the severity of the attack. Cushing[1] has shown that cases otherwise fatal may be saved by making a large osteoplastic flap over the motor region of the side opposite that paralyzed. If the clot is on the surface it can be removed, if within the brain there will be bulging, and by puncturing the cerebral substance through a fissure, the clot can sometimes be found and evacuated; whether found or not good results ensue if decompression is made. The indications for this procedure are a steadily rising blood pressure to 250 mm., and coincident with the rise a slowing of the pulse to 50 per minute. Cases presenting these conditions have generally been fatal when not operated upon.

At the end of three or four weeks the muscles may be stimulated with the faradic current and massage. The extensor muscles should only be so treated, as this tends to lessen the contracture. If contractures develop, warm baths and passive movements may be helpful. The functions of the bowels and kidneys must be regulated, and arterial tension kept low by means of nitroglycerin if necessary. The diet should be simple and easily digested, alcohol avoided, and small doses of iodide of potassium combined with tonics employed. If possible a warm, equable climate should be selected as a place of residence, and the patient should always be cautioned to avoid everything that would tend to elevate the blood pressure.

In *ingravescent* apoplexy it is important to stop the hemorrhage, for if it breaks into the ventricle, as it will eventually if not checked, death ensues. For this purpose the common carotid has been tied with success in a few instances.

If the case is one of those which sometimes occur when the

[1] Amer. Jour. Med. Sci., 1903, p. 1017.

diagnosis between hemorrhage and thrombosis or embolism cannot be made, active treatment should not be directed to either condition, but an expectant plan adopted. The treatment for hemorrhage in a thrombotic case would do harm, and *vice versa*.

Acute Softening of the Brain.—Etiology.—Acute softening is caused by the sudden plugging of a vessel by an *embolus* or *thrombus*.

Embolism occurs oftenest between the ages of twenty and fifty; and the predisposing causes are endocarditis, acute infectious diseases, profound anemia, pregnancy, and blood dyscrasias and malaria, in which the small arteries of the cortex may be blocked by the parasites and their products.

Thrombosis is most frequent between the ages of fifty and seventy. The predisposing causes are syphilitic, lead, or gouty arteritis, fatty heart, and blood dyscrasias.

Morbid Anatomy and Pathology.—Soon after the vessel is plugged, degeneration and softening of the area supplied by it begin. If a terminal artery, there may be *infarction*, either *hemorrhagic*, in which the territory is infiltrated with blood; or *anemic*, in which the area is pale, swollen, and necrotic. The nerve fibers degenerate and become fatty; the neuroglia swollen and edematous. In the cortex the softened area is at first red in color (red softening), changing to yellow (yellow softening). In the white matter the area is white in color, containing a few red punctate spots. Inflammatory changes are common in and about the softened area. The degenerated tissue is gradually absorbed; if small, a scar results; if large, a cyst.

If an embolus is derived from an infected focus, as, for instance, an ulcerative endocarditis, an abscess may result (p. 338).

In thrombosis there are usually evidences of either atheroma or syphilitic arteritis.

Secondary degenerations occur as in hemorrhage.

Symptoms.—In *embolism* the onset is usually sudden, and is characterized by a brief period of slight convulsive twitchings, followed by loss of consciousness and hemiplegia. Coma may be absent, and when present is of briefer duration

than in hemorrhage. There are either no or very slight temperature changes.

In *thrombosis* prodromes are common. They consist of headaches, vertigo, numbness of the extremities, drowsiness, and in syphilitic cases of transient paralyses, especially of the eye muscles. The onset is gradual, it taking several hours, or even longer, for the hemiplegia to develop. The patient may not lose consciousness, and if he does it is also apt to develop gradually. The attacks, however, may be sudden. They frequently occur during sleep. The temperature may fall slightly, followed by a rise; but as a rule, it is normal. The pulse is normal or possibly weak; the face is not flushed or cyanosed. In some cases there may be some increase of blood pressure due to kidney or arterial disease which existed before the attack. The resulting hemiplegia possesses the same general characteristics as that due to hemorrhage. It is apt to improve quicker, for the reason that the paralysis is usually due to plugging of the middle cerebral or its branches, and as the cortical arteries anastomose to some extent a collateral circulation is formed. In thrombosis mental symptoms similar to those occurring after hemorrhage are apt to follow. Thrombosis occurs in the *vertebral, basilar,* and *posterior cerebral arteries* oftener than does hemorrhage from them. The symptoms of plugging of a vertebral artery (see posterior cerebellar artery) are those of an acute bulbar palsy. When the basilar is occluded, the symptoms are similar to those due to pontine hemorrhage. If the main trunk of a posterior cerebral is blocked, hemianopsia with sensory aphasia will result; hemianesthesia will also be present, as this artery supplies the optic thalamus (p. 315).

Thrombosis occluding the *posterior inferior cerebellar artery* (Plate III) causes characteristic symptoms. They develop suddenly without loss of consciousness. There may or may not be slight weakness of the limbs of the side opposite the lesion which, if present, soon clears up. Pain and temperature sensations are diminished in these limbs and in the distribution of the fifth nerve of the opposite side. Spontaneous pain and paresthesia may be present in this area. Ataxia

may be present in the limbs on the side of the lesion, with a tendency to fall to that side. Paralysis of the soft palate, larynx, and muscles of deglutition is present on the side of the lesion. There is also smallness of the pupil, narrowing of the palpebral fissure and retraction of the eyeball (cervical sympathetic). The deep reflexes vary. Headache is often intense, and hiccough, rapid pulse, and vomiting may occur. Rare symptoms are diminished senses of pain and temperature on the opposite side, vertigo, tinnitus, disturbance of micturition, facial paralysis (supranuclear), weakness of external rectus, of the tongue, and diminished sense of taste on the side of the lesion.

Mills[1] believes occlusion of the *superior cerebellar artery* (Plate III) to be indicated by ataxia of limbs on same side as the lesion, loss of pain and of the power of emotional expression, temperature senses and deafness on the opposite side. Occlusion of the *anterior spinal arteries* (Plate III) at their origin from the vertebrals causes paralysis of the limbs of both sides with increased reflexes and loss of or diminished sense of position.[2]

Diagnosis.—When the coma is gradual in its onset the condition is not apt to be mistaken for any of the causes of coma mentioned under hemorrhage, excepting, possibly, that due to syphilitic meningitis. Here, and also in those cases that are sudden in onset, the same rules of diagnosis apply as were stated for hemorrhage. It is important when possible, which it sometimes is not, to distinguish between acute softening and hemorrhage. The following table will prove useful:

Hemorrhage.	Acute Softening.
Age, forty to fifty; sudden onset, with deep coma and paralysis; attack occurs after muscular exertion; early rigidity; unequal pupils; stertorous breathing; face flushed; pulse slow, full, and hard; marked increase of blood pressure; initial fall of temperature followed by a rise, more marked on the paralyzed side. Choked disk.	Age, either before thirty or after fifty; previous history of syphilis, or just preceding acute infectious disease; prodromes; gradual onset (thrombosis); attack occurs during night (thrombosis); coma often absent or transitory; heart's action weak (thrombosis) or endocarditis present (embolism) or other cause of embolism (p. 333).

[1] Jour. Nerv. and Ment. Dis., February, 1912, p. 73.
[2] Spiller, Jour. Nerv. and Ment. Dis., December, 1908, p. 775.

Encephalitis affecting the cortical cells in the motor region may simulate thrombosis. The symptoms, however, would probably follow some infectious process (p. 337).

Prognosis.—The prognosis as regards recovery from the attack is somewhat better than in hemorrhage. When due to embolism recurrence is not likely, but the reverse obtains when thrombosis is the cause. Recovery from the attack is usually more complete in softening than in hemorrhage.

Treatment.—The treatment of the attack consists of rest (the head being low), attention to the bowels and kidneys, heart stimulants and arterial dilators, as alcohol, strophanthus, nitroglycerin, and caffeine, and the general hygienic measures recommended for hemorrhage (p. 332). If a history of syphilis is obtained, vigorous antisyphilitic treatment should be instituted (p. 413).

Patients complaining of the characteristic prodromes should at once be placed on cardiac stimulants, as caffeine, strophanthus and nitroglycerin, and rest enjoined.

The treatment of the chronic stage is similar to that of hemorrhage.

ACUTE HEMORRHAGIC ENCEPHALITIS

Definition and Varieties.—This term is applied to a *symptom complex* due to foci of inflammation scattered throughout the brain and which are not accompanied by suppuration.

The gray matter of the cortex may alone be affected—the polioencephalitis of Strümpell, who believes that a certain number of cases of cerebral palsy in children are due to encephalitis localized in the motor region (p. 253).

The gray matter about the aqueduct of Sylvius, with involvement of the nuclei of the motor nerves of the eye, may be the seat of the process, the so-called polioencephalitis superior of Wernicke. The process may be confined to the gray matter of the floor of the fourth ventricle, involving the nuclei of the other motor cranial nerves, and this form is known as polioencephalitis inferior (pp. 149 and 264). The

cerebellum may also be affected. These forms may be combined.

Etiology.—The chief causes are chronic alcoholism and the acute infectious diseases, especially influenza. Cases have also been attributed to poisoning by lead and ptomains, and to trauma, the latter possibly acting by lowering the resisting power to pyogenic organisms. It is more common in children and young adults.

Morbid Anatomy.—Scattered throughout the brain are foci of inflammation, characterized by hyperemia, hemorrhage, serous exudation, and round-celled infiltration. Suppuration never occurs. They appear as red or yellow points. The small bloodvessels have degenerated walls and are surrounded by blood corpuscles and round cells. The nerve cells are swollen and may finally degenerate. There is also more or less degeneration of various tracts in the brain, notably the motor. The bloodvessels are probably the starting point of the process, the changes being similar to those which occur in acute poliomyelitis, which may coexist.

Symptoms.—The symptoms of polioencephalitis superior and inferior having already been discussed (pp. 149 and 264), those due to a combination of these and Strümpell's form only are here described.

There may be a prolonged stage of headache, vertigo, and general malaise; or it may begin suddenly with vomiting, restlessness, stupor, and slight rigidity of the neck. The temperature may either not rise much above normal, or there may be an initial chill followed by high fever. Respiration is usually rapid. Leukocytosis is present.

The focal symptoms depend upon the location of the lesions. They do not all develop at the same time, but appear at intervals as the disease progressively attacks different regions. There may be paralysis of some or all of the ocular muscles, optic neuritis, paralysis of the muscles of the face, throat, tongue, choreiform movements of the limbs, paralysis of a hemiplegic or monoplegic type, hemianesthesia, ataxia or hemiataxia, conjugate deviation of the eyes and aphasia. Epileptiform seizures of the Jacksonian type have been

observed.[1] Of course all of these symptoms are not present in every case, but they occur in different combinations in different cases. Symptoms of acute poliomyelitis may also be present.

Diagnosis.—The disease may be mistaken for meningitis. It differs from this in the more sudden onset of stupor, absence of markedly contracted pupils, hyperesthesia, and great muscular rigidity. Examination of the cerebrospinal fluid in encephalitis will show a clear or slightly turbid sterile fluid and a large increase of lymphocytes (p. 97). The diagnosis may, however, be very difficult. The symptoms described following infectious disease or occurring in a chronic alcoholic, would point to encephalitis. Cerebral syphilis is distinguished usually by more gradual onset and other evidences of the disease.

Prognosis.—The prognosis is grave; but cases, especially those following infectious diseases, recover. Those which do not recover have often some permanent paralysis remaining.

Treatment.—Treatment consists of purgation with calomel and application of ice-bags to the head. Hexamethylenamin should be given. If there is much restlessness, bromides or other sedatives are indicated. If the patient is debilitated, stimulants should be given. Lumbar puncture has been advised where there is much intracranial pressure. In the period of convalescence, tonics and treatment to restore the paralyzed muscles.

Acute Suppurative Encephalitis (Abscess).—Etiology.—Suppuration of the brain substance practically always is due either to extension of inflammation from neighboring parts or infection from a distance through the blood. The chief causes are in the order of their importance:

1. Extension from disease of the middle-ear or mastoid cells, which may be by passing of the infection from the roof of the mastoid antrum to the sigmoid sinus, where an infective thrombosis is produced; by involvement of the dura and the formation of a subdural abscess which extends

[1] Mills, Review of Neurology and Psychiatry, February, 1907, p. 89.

to the arachnoid and pia mater; by extension of inflammation along the lymph spaces or thrombosed veins into the brain substance.

2. Traumatism to the head, with or without abrasion of the skin, and usually associated with fracture of the skull.

3. Remote septic processes, as ulcerative endocarditis, tuberculosis of the lungs, fetid bronchitis, empyema, suppuration of the liver, localized caries of the ethmoid, nasal bones, and of the orbit; and pyemia.

4. The infectious fevers, especially influenza, are occasional causes.

Most cases occur between ten and thirty. Males are more frequently attacked than females.

Morbid Anatomy.—The abscess may be solitary or multiple, more frequently the former, and may be produced by any one of the pyogenic bacteria. In acute cases the suppuration is not limited, but in chronic cases the abscess is enclosed in a capsule. In acute cases the pus is reddish in color and is mixed with softened brain matter. In the chronic cases it is of greenish tint, acid reaction, and peculiar odor, resembling somewhat that of sulphuretted hydrogen. The brain substance about the abscess is usually edematous. The size varies from that of a walnut to that of an orange. Abscesses occur most frequently in the cerebrum, usually on the right side, and in either the temporal or the frontal lobe. Those due to ear disease are nearly always either in the temporal lobe or cerebellum.

Symptoms.—Abscesses of the brain may run either a rapid or a slow course. The *former* usually follow trauma, and the symptoms may be general or focal. The *general* symptoms are, following an injury, severe headache, vomiting rigors, irregular fever, mental dulness, and delirium. If there are no focal symptoms, which is sometimes the case, the condition may be mistaken for meningitis. When *they are present* they consist of paralysis, usually of the hemiplegic type; aphasia, epileptiform convulsions, and, if in the cerebellum, cerebellar incoördination. Optic neuritis may occur. The pulse is usually slow, 60 to 70. The mental dulness is

succeeded by coma, and death occurs from exhaustion in from a few days to a month after the onset.

The *chronic form* most commonly follows ear disease. After the abscess has formed the symptoms may remain practically latent for weeks, months, or even years. During this period the patient may suffer from headache, vertigo, and mental depression. Occasionally there may be acute exacerbations associated with severe headache, vomiting, and delirium or convulsions, after which the patient returns to his former state of health. The temperature is normal or subnormal. When the terminal stage sets in, either symptoms similar to those of the acute form described above appear, or there is a sudden epileptic seizure or a rapid development of coma, in which death soon occurs.

Phlebitis, with consequent *thrombosis*, of the superior petrosal and lateral sinuses may coexist, especially in those cases due to ear disease. When present, edema about the ear and neck and hardness of the jugular veins will be manifest. Meningitis may also be present, and would usually be indicated by rigidity of the neck, hyperesthesia, and, possibly, paralysis of the cranial nerves.

Diagnosis.—The diagnosis of abscess of the brain is often difficult. It is based upon the occurrence of the symptoms as detailed above, coming on after head injury or the previous occurrence of aural or nasal disease, or any of the causes mentioned under etiology.

In abscess the pulse is slow; an increase in the pulse rate coincident with elevation of temperature makes its diagnosis doubtful, although this may occur if meningitis develops, as it may do. In acute abscess leukocytosis will be present.

Phlebitis and consequent thrombosis of the lateral and petrosal sinuses may occur independently of abscess. Its presence would be distinguished by the symptoms mentioned above.

The *location* of the abscess is based upon the presence of symptoms pointing to interference with the functions of that part of the brain where the abscess is situated. (See Cerebral Localization.) They often occur, however, in

silent regions, in which case localizing symptoms would be absent.

The diagnosis may have to be made from tumor and meningitis (pp. 221 and 360). The differential points are given under the description of those diseases. Meningitis may, however, be associated with abscess.

Prognosis and Treatment.—The *prognosis* is bad. Cases do recover if surgical interference is resorted to in time. In a few instances evacuation has taken place spontaneously through the nasal passages, but the majority die.

The *treatment* is opening the skull over the seat of the abscess and evacuating it.

TUMORS OF THE BRAIN

Etiology.—In a small percentage of cases a history of a blow or fall upon the head has been obtained. Males are more subject to brain tumor than females. They occur in persons of all ages.

Varieties.—Almost every known variety of tumor has been found in the brain. Certain ones are common, while others are rare. Those which are common, in the order of frequency, are tuberculous tumors, sarcoma, glioma, gliosarcoma, cyst, carcinoma, and gumma. The rare ones are fibroma, angioma, myxoma, osteoma, lipoma, psammoma, cholesteatoma, echinococcus cyst and circumscribed serous meningitis (p. 227).

Tuberculous tumors are most common in childhood; they may be primary, but are usually secondary to tuberculous disease elsewhere. They are often (20 per cent.) multiple. Their size varies from a small collection of miliary tubercles lying in a mass of thickened pia mater to a large, solid, circumscribed mass, with a hard, cheesy, or broken-down granular centre and a distinct capsule. They may be found within the cerebral tissue at some distance from the surface, but are usually found in connection with the meninges and large cerebral vessels: hence are upon the surface or at the base. Tuberculous meningitis may coexist.

Sarcoma rarely occurs secondarily to sarcoma elsewhere in the body. Frequently there may be more than one present. The tumor is usually encapsulated, hard in consistency, and is easily separated from the brain substance, which it rarely infiltrates; that it does so occasionally must be remembered. It grows rapidly and is not vascular. Any part of the brain may be the seat of sarcoma, but it is more frequent in the cortex and cerebellum.

Glioma is usually primary. They are softer in consistency than sarcoma and usually infiltrate the brain substance. They have a marked tendency to become cystic. They may appear in any part of the brain, but are most frequent in the white matter. As a glioma grows, it destroys the nerve tissue and does not compress it as sarcoma does. It is a vascular tumor, and renders the patient liable to apoplectiform attacks.

Gliosarcoma partakes of the nature both of sarcoma and glioma. It is often cystic, and is not encapsulated. The symptoms resemble those of glioma.

Carcinoma is usually secondary to carcinoma elsewhere. It is most common in adults above the age of fifty. It grows rapidly, infiltrates the brain substance, and is very vascular.

Cystic tumors may be the result of circumscribed serous meningitis (p. 227) of cystic degeneration occurring in a glioma or gliosarcoma, or as the result of infection with the echinococcus. The latter are rare in this country.

Gumma is rare in childhood. It does not occur as the result of inherited syphilis. It may occur any time from one to twenty years or more after the primary infection. Gumma may occur either as a soft, gelatinous mass anywhere in the membranes, most commonly at the base; or as a harder, circumscribed tumor in the meninges, usually involving the cortex or cerebellum. The growth is rapid.

The **remaining varieties** of tumor are of great rarity; they may occur in either children or adults.

A brain **in which tumor has developed** usually presents the following characteristics: The membranes are tense, owing to pressure, the convolutions are flattened, and in the region of the growth do not pulsate, the ventricles are distended

with serous fluid, the brain tissue is wet, the weight of the brain is increased, and frequently there will be found about the tumor a zone of softening.

Symptoms.—The symptoms may be divided into two classes:

1. *General symptoms*, which occur independently of the position of the tumor.

2. *Focal symptoms*, or those dependent upon interference with the functions of the cerebral centres or of the tracts which run to or from them.

General Symptoms.—These depend upon the rapidity of growth, vascularity, and variety of the tumor. They vary in severity from time to time. For instance, while the tumor is growing rapidly they may be severe; if it becomes stationary for a time, they may almost disappear. They are also influenced by agents which produce a temporary cerebral anemia or hyperemia. Such are headache, general convulsions, papilledema, reversal and interlacing of the color fields, secondary optic atrophy, changes in the disposition and mental power, vomiting, vertigo, insomnia, changes in the pulse rate, attacks of syncope, apoplectiform attacks, polyuria, and progressive emaciation.

Headache is a most constant symptom; it is usually intense. It may be either dull, heavy, and continuous, with sharp paroxysms; or it may be intermittent. It is increased by physical or mental strain or emotional excitement. If gumma is present, it is usually worse at night. In addition to the pain, there may be indefinite sensations, as fulness, pressure, tightness as if a band were about the head. The location of the pain is usually frontal or occipital, and rarely has any relation to the location of the tumor.

The *causes* of the headache are increase of the intracranial pressure and consequent stretching of the membranes, or the direct involvement of the dura and consequent irritation of the branches of the fifth nerve.

General convulsions are particularly liable to occur as an early symptom in children, but may be the first symptom in adults. They may occur at intervals for some time previous to the development of other symptoms, and are of an

epileptiform nature. Special attention should be paid to the location and nature of the aura and the parts where the spasmodic movements begin. (See Jacksonian Epilepsy.)

The occurrence of general convulsions is indicative of rapid growth of the tumor, and has no localizing value. Death frequently occurs in a convulsion.

Changes of disposition and mental power occur with considerable frequency in the course of brain tumor independently of the situation, but are more marked in those of the frontal lobes. Children become fretful and irritable, refuse to play and lose interest in things which formerly attracted them. Often they prefer to keep quiet and secluded, and may become somnolent and lethargic. In adults the individual loses interest in his business and surroundings; he becomes apathetic, and may sit in one place for hours doing nothing; if aroused, he may answer questions intelligently; finally, however, as the case progresses, the memory is lost and dementia appears. Attacks of maniacal excitement may occur and delusions develop. Any of the manifestations of hysteria may occur.

Papilledema and Atrophy.—Papilledema or choked disk is a most important symptom (p. 137). A severe form may exist without defect of sight; hence the eye-ground should always be examined in all cases presenting cerebral symptoms.

Optic atrophy, if found, is the result of a previous papilledema, which occurs in about 80 per cent. of brain tumors, and is more frequent in tumors of the base and in the region of the basal ganglia and in the cerebellum than in those of the cortex and centrum ovale. According to Marcus Gunn,[1] the condition of the optic nerves may aid in the localization of a tumor to the following extent:

1. An intense double "papilledema" with much swelling and surrounding retinal change, coming on quickly, suggests the cerebellum.

2. One-sided "papilledema" or marked difference suggests the cerebrum, and is, on the whole, in favor of the tumor

[1] Brain, 1898, p. 337.

being on the same side as the excess of neuritis, when there are other reasons for localizing one in the front of the cerebrum.

Visual Fields. An important and early symptom is reversal and interlacing of the visual fields. This symptom was formerly believed to be only present in hysteria (p. 466), but Cushing and Bordley have shown that it is a frequent symptom of tumor.[1]

Vomiting is rather more common in children. It may or may not be accompanied by nausea, and occurs independently of the taking of food. Attacks are frequent in the morning just before rising, and often at such times the slightest movement of the head is sufficient to provoke a paroxysm.

Vertigo may occur with or independently of vomiting. It is often excited by changes in position, and the attacks are paroxysmal. It is most common with tumors in the posterior fossa, cerebellum, pons, or at the base involving the auditory nerve.

Insomnia is most frequently complained of by adults, especially those suffering from syphilitic growths.

Syncope is sometimes an occasional symptom in tumors of the posterior fossa.

Polyuria, with or without glycosuria, may develop in the course of brain tumor as a symptom of increased pressure. It has also occurred in small tumors of the medulla and cerebellum probably due to irritation of the nucleus of the vagus.

Menstrual Disturbances.—Amenorrhea may be an early symptom in women, especially in tumor of the hypophysis.

Focal Symptoms.—These depend upon the location of the tumor; when it is situated in one of the so-called "silent regions" of the brain they are practically absent. Focal symptoms are due, first, to the irritation or destruction of the centre in which it is situated, and, second, to either irritation of neighboring centres or to loss of their functions due to pressure. Thus a tumor situated so as to destroy

[1] Archives of Ophthal, November, 1909, p. 451.

the left third frontal convolution, producing aphasia, would also in time, by the pressure it excited upon them, destroy the functions of the motor centres at the foot of the central convolutions. Dr. James Collier[1] has called attention to several sources of error which may arise in interpreting supposed localizing symptoms. He shows: (1) That local signs appearing *late* in the course of intracranial tumor, when general signs *alone* have preëxisted, are often of false portent; (2) that in such cases the local signs have often been due to the presence of independent, and later developed, vascular lesions, meningitis, hydrocephalus (developed secondarily to the tumor), which is often a cause of Jacksonian fits, when they occur late in the disease; local spreading edema of the brain (localized edema in the region of a tumor is frequent); secondary deposits of newgrowth and posterior degeneration; (3) that the absence of usually accepted local signs during the early days of illness in intracranial tumors is in itself an important localizing sign, confining the disease to the supratentorial region; (4) that true localizing signs, at one time present, may later become concealed or undemonstrable, owing to the development of other signs, and that in cases which come under observation for the first time, late in the disease, diagnosis may be difficult, erroneous, or impossible.

Therefore, in endeavoring to localize a growth, attention must be paid to the sequence in which the symptoms have developed, not too much stress being laid upon those which appear late. The reader is referred to Cerebral Localization for information regarding the situation and functions of the cortical centres, and the functions of the basal ganglia, pons, medulla, and cerebellum. Fig. 5 shows the course and relations of the projection fibers, and Fig. 97 the situation and relations of the cranial nerves at the base of the brain.

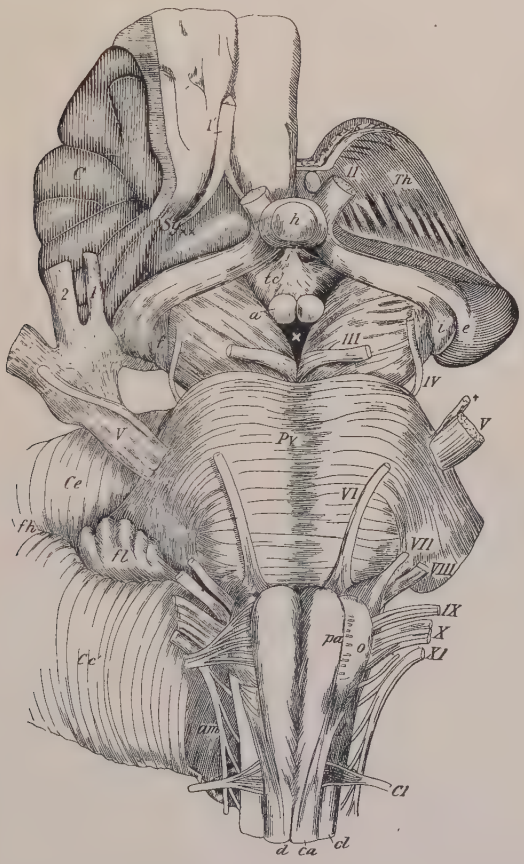
A knowledge of the situation and anatomical relations of these centres, tracts, and nerves is essential to interpret properly focal symptoms.

A **slow-growing tumor** which does not infiltrate, but gradually compresses the brain, may not cause such symptoms

[1] Brain, 1904, p. 490.

until it has reached large size, for nervous tissues often accustom themselves to gradually exerted pressure.

FIG. 97



The base of the brain and the cranial nerves, crura, pons, and medulla: *I* to *XII*, the cranial nerves; *Th*, optic thalamus; *h*, pituitary body; *tc*, tuber cinereum; *a*, corpora albicantia; *P*, pes pedunculi; *i*, interior, and *e*, exterior, geniculate body; *PV*, pons Varolii; *pa*, anterior pyramid of medulla; *o*, olive; *d*, decussation of anterior pyramids; *ca*, anterior column of spinal cord; *cl*, lateral column of spinal cord; *Ce*, cerebellum; *fl*, flocculus of cerebellum; (Allen Thompson.)

The usual combination of focal symptoms in the more commonly affected parts of the brain are as follows:

Frontal Lobes.—The focal symptoms when the cortex of the frontal lobes is involved are principally mental and resemble those of paresis. They are an undue tendency to joke (witzelsucht), mental inactivity, lack of judgment, change in character, irritability, forgetfulness, inability to concentrate the attention and to think continuously. T. Grainger Stewart[1] has called attention to two symptoms which he states are characteristic of tumors in this location. They are absence, diminution, or easy exhaustion of the abdominal reflex on the side opposite the tumor, and the occurrence of a fine, rapid, vibratory tremor in the limb of the same side as the growth. A staggering gait, similar to that of cerebellar disease, has also been obserevd. Kennedy[2] states that an important symptom is the finding of a true retrobulbar neuritis with the formation of a central scotoma and primary optic atrophy on the side of the lesion, together with concomitant papilledema in the opposite eye. Mental symptoms are not present in growths lying upon the base of the frontal lobe where it lies on the orbital bone. There may be loss of the sense of smell, which, if one-sided, is probably on the side of the tumor. Tumors involving the left side are apt to cause a degree of aphasia more or less marked. Localized spasms and paralysis may also be present. If on the left side motor apraxia (p. 309) may be present.

Motor Region.—Cortical tumors cause either tonic or clonic convulsions, occurring at intervals. These may be either limited to one part of the body or extend from the part first affected to other parts (Jacksonian Epilepsy) (see p. 439). The starting point of the spasm indicates the situation of the growth. Such spasms are usually followed by a temporary paralysis, which, as the tumor grows, becomes permanent. The spasm may be preceded by tingling and numbness. Permanent paresthesia indicates a tumor behind the fissure of Rolando. Tumors situated beneath the cortex of the motor region (subcortical) usually cause paralysis without

[1] Rev. Neurol. and Psychiat., December, 1906, p. 809.
[2] Amer. Jour. Med. Sci., September, 1911, p. 355.

spasm; or, if spasm finally occurs, owing to extension of the growth to the cortex, it is preceded by paralysis. Slight degrees of anesthesia may be found.

Parietal Region.—Tumors in this region may cause disturbances of sensation and muscle sense, as shown by ataxia, in the limbs of the opposite side. Astereognosis is also present. Disturbances of common sensibility would indicate a lesion of the ascending parietal convolution. A lesion there could, of course, cause ataxia and astereognosis also, by making pressure on the superior and inferior parietal lobules. Word-blindness is due to lesion of the left inferior parietal lobe (angular and supramarginal gyri), as is also homonymous hemianopsia.

Occipital Region.—Tumors here produce lateral homonymous hemianopsia, in which Wernicke's pupillary inaction sign is not present (p. 65). Visual hallucinations such as flashes of light may occur. They usually are seen on the side opposite the tumor.

If left sided in right-handed people, and *vice versa* (see Aphasia), there may also be a condition known as mind-blindness, in which there is incapacity to understand the nature of things seen and formerly known (agnosia, p. 309). If the lesion extends into the parietal lobe, hemianesthesia, hemiataxia, and some hemiplegia may result.

Temporal Lobe.—Growths involving the first and second temporal convolutions of the left side (in a right-handed person) causes word-deafness. Tumors in other parts of this lobe produce no definite symptoms, those present being due to pressure upon the motor tract or the temporal convolutions above mentioned. If the uncinate gyrus is involved the peculiar attacks described on p. 437 may occur (uncinate group of fits).

Tumors of the corpus callosum are rare. They cause mental symptoms similar to those produced by involvement of the frontal lobes, often followed by a double hemiplegia, one side being affected before the other; motor apraxia (p. 309) is frequently found.

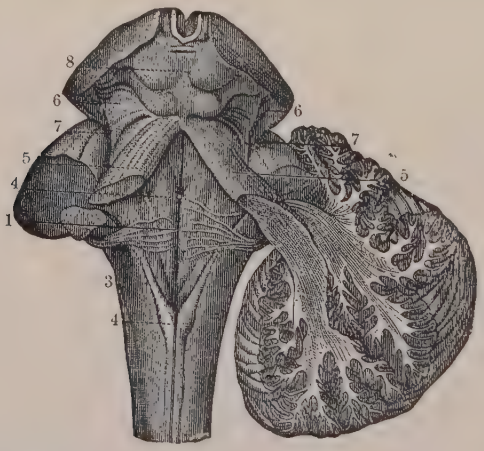
Tumors of the corpus striatum may cause pressure upon either the motor or the sensory tracts, usually the motor,

passing through the internal capsule. Other symptoms of disease affecting it are detailed (pp. 248 and 302).

In **tumors of the optic thalamus** there may be mental stupidity, nystagmus, and weakness of ocular movements on the side of the tumor.

Roussy states that the following symptoms constitute the thalamic syndrome; hemianesthesia, involving deep sensibility more than the superficial forms, with persistent, paroxysmal, spontaneous pains in the affected side; little or

FIG. 98



The three peduncles of the cerebellum: 1, rhomboidal fossa of the fourth ventricle; 2, striæ acusticæ; 3, inferior peduncles or restiform bodies; 4, columns of Goll; 5, superior cerebellar peduncles; 6, lemniscus; 7, middle peduncles; 8, corpora quadrigemina. (From Hirschfeld and Sappey.)

no hemiplegia; absence of Babinski reflex; hemiataxia and frequently irregular involuntary movements of the athetoid, or choreic type, in the affected limbs. A tumor may make pressure on neighboring tracts and centres, and hence other symptoms as more or less hemiplegia and astereognosis may be present. Other symptoms noted have been hemianopsia, causeless laughter and crying, inability to move the facial muscles in expressing the emotions (p. 302), with preservation of the power of making voluntary movements.

Tumors of the corpora quadrigemina, owing to their proximity to the cerebellum, cause disorders in gait similar to cerebellar disease (Fig. 98), and paresis of the motor nerves of the eye (Fig. 38), the usual type being that of ophthalmoplegia externa, and loss of associated movements of the eyes upward. There may also be hemianopsia of similar

FIG. 99

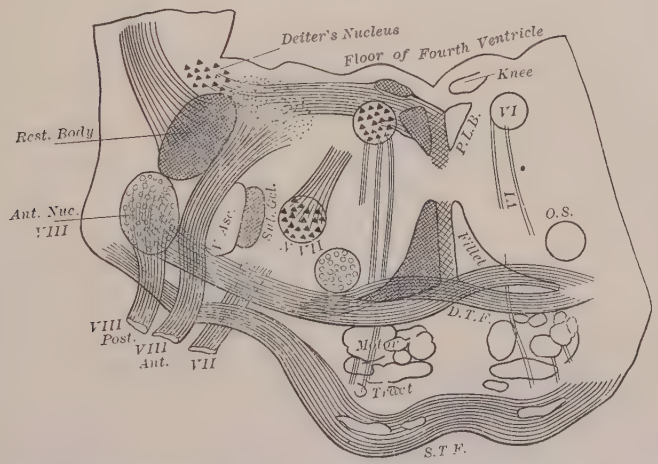


Diagram of section through lower part of pons. This diagram is not quite accurate, as it represents, for convenience, certain structures which are not actually observed at exactly the same level. (Thus it shows the origin, course, and exit of the seventh nerve.) *VI*, abducens nerve and nucleus; *N. VII*, nucleus of facial nerve; *VII*, facial nerve; *Ant. Nuc. VIII*, anterior nucleus of auditory nerve; *VIII Ant.* and *VIII Post.*, anterior and posterior or deep and superficial roots of the auditory nerve; *Corp. Rest.*, restiform body; *Knee*, knee of the facial nerve; *O. S.*, superior olive; *P. L. B.*, posterior longitudinal bundle; *S. T. F.*, superficial transverse fibers of the pons; *D. T. F.*, deep transverse fibers of the pons; *V. Asc.*, ascending root of trigeminus; *Sub. Gel.*, substantia gelatinosa. The internal or "chief" auditory nucleus lies just internal to the nucleus of Deiters.

type to that occurring in growths involving the thalamus. There may also be some dulness of hearing, more marked in the ear opposite the lesion. Weakness of the contralateral limbs may be present. (See also Tumors of the Cerebellum.)

A **tumor involving the crus** would cause hemiplegia of the

opposite side, with complete third-nerve paralysis upon the same side as the lesion (Fig. 86). Hemianopsia may also occur if pressure is made on the optic tract. If extension occurs into the *tegmentum*, hemianesthesia will coexist.

Tumors of the pons produce symptoms according to their size and location. If situated high up, a crossed paralysis similar to that produced by a lesion of the crus would result.

FIG. 100

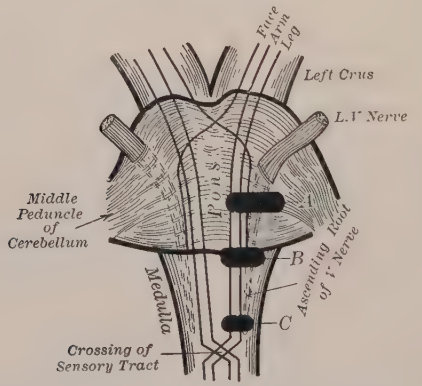


Diagram illustrating the mechanism of crossed anesthesia. Any lesion interrupting the general sensory tract from the lines above the crossing of this tract in the medulla and the ascending root of the fifth nerve on the same side will produce crossed anesthesia. The conditions will vary, however, with the level of the lesion. Thus, a lesion at *A* will cause typical crossed anesthesia and rotation of the body to the side of the lesion from involvement of the middle peduncle of the cerebellum; a lesion at *B* will cause complete anesthesia of the opposite limbs and trunk, but the anesthesia of the face on the same side as the lesion will not involve the entire face, sensation remaining normal in its upper portion; a lesion at *C* will cause anesthesia of the entire opposite half of the body, but *probably* only partial and greatly limited anesthesia of the face on the same side as the lesion. (After Starr.)

If a little lower down, motor and sensory paralysis of the arm and leg on the opposite side, with paralysis (sensory portion) of the fifth nerve on the same side as the lesion. In the lower part hemiplegia upon the opposite side, with paralysis of the seventh nerve upon the side of the lesion. Also in this location the sixth and eighth nerves may be involved with the seventh (Fig. 99). Crossed hemianesthesia may be associated with the hemiplegia, and varies in degree

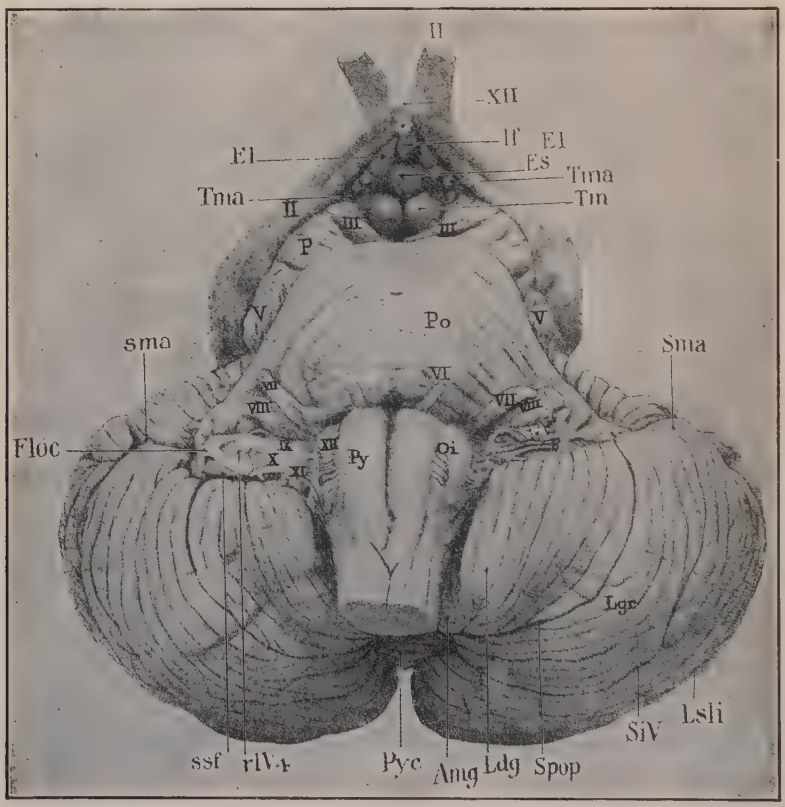
of completeness according to the location of the lesion (Fig. 100). Dissociation of sensation has been observed. If on the lateral edge involving the peduncles, forced movements and ataxia will result. There may also be, if the sixth nucleus is involved, conjugate deviation of the eyes, in which the eyes look away from the side of the lesion (Fig. 39, *B*). There may also, if this or the posterior longitudinal fasciculus is involved, be loss of associated lateral movements toward the side of the lesion, while the power of convergence remains.[1] (See also Tumors of Cerebellum.) The knee-jerks are frequently absent. Tumor of the *medulla*, if large, would produce symptoms of progressive bulbar palsy, plus more or less hemiplegia and hemianesthesia. If a small growth, crossed paralysis of the hypoglossal nerve with hemiplegia may occur (Fig. 86).

Tumors of the Cerebellum.—Tumors in this location are quite frequent, both in children and adults. They often cause hydrocephalus, as they are apt to obstruct the foramen of Majendie (p. 230). The symptoms of this condition, therefore, are often combined with those due to the cerebellar disease. They may be situated either in the middle or one of the lateral lobes. Tumors in the cerebellopontile angle (Fig. 101) cause symptoms sometimes so closely resembling cerebellar tumors that they will also be discussed here. The relations of the cerebellum are also shown in Fig. 5. In view of the possibility of the successful removal of growths in this region, especially those in the lateral lobes, by operation, their diagnosis and accurate localization are of the utmost importance. When the *middle lobe* is alone affected, the main symptoms, in addition to the early appearance and severe character of the papilledema and headache, which is usually occipital, are cerebellar ataxia, vertigo, a tendency to fall, usually backward, nystagmus, the Romberg symptom, weakness or asthenia of the muscles of the back, and sometimes of those of the legs. This asthenia has been thought by many to be the cause of the ataxia. Occasionally rigidity and retraction of the

[1] Potts and Spiller, Univ. of Penna. Med. Bull., December, 1903.

head, even to the extent of opisthotonos and tetanic-like seizures, have been observed.[1] The knee-jerks may be

FIG. 101



The base of the brain, the cranial nerves, and the cerebellum: *II*, optic nerves; XII, optic chiasm; *lf*, infundibulum; *El*, lateral part of tuber cinereum; *Es*, tuber cinereum; *Tm*, mammillary bodies; *P*, crus cerebri; *Po*, pons; *Py*, pyramid of medulla; *Oi*, olivary body; *sma*, transverse fissure of cerebellum; *floc*, flocculus; *ssf*, subfloc. fissure; *rlV4*, diverticulum of fourth ventricle; *Pyc*, pyramid of Malacarne; *Amg*, amygdalus; *Ldg*, digastric lobe; *Lgr*, slender lobe; *Lsli*, semilunar lobe; *II* to *XII*, cranial nerves; *CPA*, cerebellopontile angle. (Dejerine.)

[1] Jackson, Brain, 1906, p. 425.

increased, normal or absent, and they may vary to this extent in the same patient. The Babinski reflex is not present in uncomplicated cerebellar disease. Palsies of ocular muscles, sometimes transient, frequently occur in cerebellar tumor; the most frequent is that of the sixth nerve, which, in middle-lobe lesions, is apt to be bilateral. They are due to compression of the floor of the fourth ventricle, and if they are severe, of the pons. Other ocular muscles may also be involved, especially if the growth extends to the superior peduncles, and thus involves the quadrigeminal region. Another ocular symptom sometimes seen, and indicative of involvement of the middle lobe, is what has been termed "skew deviation," *i. e.*, one eye being directed upward and inward and the other downward and outward.

A **tumor of the lateral lobe**, if confined to the outer part, may give no localizing symptoms. It is important, although sometimes difficult, to determine which side is involved, for, as has been said, growths here situated may be sometimes removed. If it involves also the middle lobe, symptoms are produced. Then, as in tumors affecting the middle lobe, ataxia of the cerebellar type and vertigo are prominent symptoms. The tendency of the patient in most cases being to fall toward the side of the lesion.[1] During vertiginous attacks external objects move from the side of the lesion to the opposite side, the rotation of the body (subjective vertigo) being in the same direction.[2] The headache, if occipital, may be more pronounced on the side of the lesion. Cranial-nerve palsies, *i. e.*, third, fourth, fifth, sixth, seventh, eighth, are more apt to occur on the side of the lesion; of these the sixth and seventh are most usually affected. In the case of the latter it is usually slight. The face also may be involved on the opposite side, in which event it is due to pressure upon the upper part of the pons, and is of the central type (p. 164). Twitchings of the face upon the side of the lesion have also been occasionally observed. Weakness or asthenia of the limbs and truncal muscles upon the side of the lesion is also a prominent symp-

[1] It has been asserted by some that the opposite is the case, but the weight of authority and experience is in favor of the statement above made.

[2] Stewart and Holmes, Brain, 1904, p. 525.

tom; the muscles are flaccid. Ataxia of the upper limb, as shown by the finger-to-nose test, is greater on the side of the lesion. Hemiasynergia and "diadococinesia," described by Babinski, may serve as corroborative signs, but are not constant. The former consists in inability to straighten the leg, after the thigh has been flexed on the body and the leg on the thigh, with one movement, it benig done in a jerky, asynchronous manner. The latter consists in the inability to perform, accurately, rapid alternate movements, as pronating and supinating the arm in rapid succession. These symptoms occur on the homolateral side. Another confirmatory symptom, if present, is that described by Batten,[1] which consists in the head being held inclined to the shoulder of the side opposite the lesion, and the face turned up to the side of the lesion. It may be present when the cerebellum is not affected. Tremor is not present if hydrocephalus has not occurred. The reflexes are as in disease of the middle lobe. *Tumors of the cerebellopontile angle* are usually encapsulated fibromata, and originally are attached to either the auditory or trigeminal nerves, usually the former. Outside of the symptoms produced by involvement of these nerves, other symptoms caused by them are due to pressure. From their location they may be confounded with either cerebellar or pontile tumors, usually the former, as they press upon both of these structures. The early symptoms are usually those of involvement of a cranial nerve, usually either the auditory or trigeminal. If the former, there is tinnitus aurium, failure of hearing, and attacks of vertigo resembling Ménière's syndrome (p. 114). Dana[2] states that these attacks are characterized by:

1. Loud, high-pitched tinnitus, or roaring and crackling noises, suddenly increased in intensity.

2. Vertigo, usually objective, and with or without forced movements.

3. A tendency to drop or fall, in one direction or another, instantly to the ground.

4. Sometimes sudden blindness and loss of consciousness.

5. In severe attacks, tonic spasms, generally of an extensor type. This lasts from one to ten minutes.

[1] Brain, 1903, p. 71. [2] New York Med. Jour., February 11, 1905, p. 270.

He states that the sudden falling, forced movements, loss of consciousness, and tonic spasm differentiate these attacks from pure labyrinthine disease. Furthermore, symptoms of involvement of the pons, medulla, cerebellum, and other cranial nerves sooner or later appear, as well as the general symptoms (headache, papilledema, etc.) of brain tumor. The former symptoms may consist of ataxia, with a tendency to fall to the side of the growth, nystagmus, irregularities in the size and reaction of the pupils, dysarthria, paralyses of the trigeminal, facial, and abducens nerves, of the extremities, soft palate, and tongue, as well as various respiratory, vasomotor, and circulatory phenomena. If the growth start in the trigeminal nerve the early symptoms, instead of the tinnitus, etc., are neuralgic pains in the course of the nerve. In many cases symptoms of bulbar involvement are absent. The following are given by Stewart and Holmes[1] as the distinguishing points between these and intracerebellar growths: Early appearance of headache and optic neuritis in the latter; greater involvement of the cranial nerves, notably the sixth, seventh, and eighth in the former (Fig. 101). The character of the vertigo in extracerebellar growths differs from that of intracerebellar growths (p. 355) in that the subjective rotation is from the opposite side to the side of the lesion. Hypotonia and paresis of the extremities on the side of the lesion is not marked in extracerebellar growths, but a spastic paralysis of the opposite side with the Babinski reflex is apt to occur, which is not the case in intracerebellar growths. Cerebellar tumors, especially when located in either the lateral lobe or cerebellopontile angle, may have to be differentiated from intrapontile growths. In most cases this can be done by observing in the latter a less degree of headache and optic neuritis and the early and greater involvement of the motor and sensory tracts (p. 352).

Henschen[2] states that in cerebellopontile angle growths which arise from the auditory nerve a radiograph will show an enlargement of the internal auditory meatus.

Tumors involving the superior cerebellar peduncles may resemble those of the corpora quadrigemina to such an extent that the distinction cannot be made.

[1] Loc cit. [2] Abstract in Rev. Neurol. and Psychiat., April, 1912, p. 186.

Symptoms and signs.	Lateral cerebellar tumors.	Extracerebellar tumors.	Intrapontine tumors.
Optic neuritis	Early and intense	Variable	Often absent or late.
Vertigo	Subjective rotation of self from the side of the lesion.	Subjective rotation of self to the side of the lesion.	Indefinite.
Cranial nerves V	Rarely affected	Often affected	Affection of these nerves often bilateral. Paresis may be supranuclear or nuclear, and grouped according to nuclear arrangement. Paralysis of a nerve on one side and of an adjacent or distant nerve on the opposite side. Permanent paralysis of conjugate deviation of the eyes.
" VI	Weakness of conjugate deviation to side of lesion. Weakness of external rectus on side of lesion. Slow deliberate nystagmus to side of lesion.	Same as in unilateral cerebellar tumors.	
" VII	Paresis slight if present.	Paresis more marked.	
" VIII	Deafness on side of lesion incomplete and variable. Tinnitus general.	Deafness on side of lesion marked — generally complete. Tinnitus referred to ear on side of lesion.	
" IX	Never affected.	Occasional paresis on side of lesion.	
" X	Never affected.	Occasional paresis on side of lesion.	
" XI	Never affected.	Occasional paresis on side of lesion.	
" XII	Never affected.	Supranuclear paresis on contralateral side.	
Motor system	Homolateral paresis, ataxia, and atonia.	Homolateral paresis and ataxia; contralateral spastic paresis common—occasionally bilateral.	Paresis often bilateral, with spasticity. Ataxia general.
Sensory system.	No change.	No change.	Occasionally hemianesthesia.
Reflexes—Tendon	Variable, often diminished.	Generally increased, especially on contralateral side.	Increased often unequally.
Superficial	Normal	Often diminished on contralateral side.	Diminished, often unequally
Plantar	Flexor	Flexor or extensor. Extensor on contralateral or both sides.	Extensor on one or both sides.
Sphincters	Not affected.	Rarely affected.	Generally affected.

—From Steward and Holmes, Brain, 1904, p. 549.

Tumors of the lateral lobe, if deeply situated, have simulated those of the superior parietal lobule.[1] The presence of nystagmus, and severe vertigo, and the absence of astereognosis, and disturbances of muscular and cutaneous sensibility, favor the former location; also, they may need to be differentiated from prefrontal growths. The peculiar mental symptoms (p. 348) and frequent later occurrence of some form of aphasia and of hemiplegia, with possibly local spasms, distinguish frontal growths.

Tumors of the base of the brain compress one or more of the cranial nerves either unilaterally or bilaterally, according as the tumor is situated on one side or in the median line; the location can be usually determined by noticing the order in which the nerves become involved; their relations to each other, and other structures at the base of the brain as indicated in Fig. 97. Optic neuritis and headache are apt to be intense. Tumors of the *Gasserian ganglion* cause intense pain and loss of or diminished sensation in the distribution of the fifth nerve. Symptoms simulating basal growths may occur in tumors situated above the tentorium. Paralysis of cranial nerves, especially the sixth, occurs in such growths, but appears late. They may be due, in the case of the sixth, to pressure of the brain downward into the foramen magnum.[2]

Tumors which destroy the *hypophysis* may produce the symptoms of acromegaly with either bitemporal or homonymous hemianopsia or *primary* optic atrophy. A number of cases have been reported in which the symptoms were primary optic atrophy and lack of development of the sexual apparatus, and in the case of females, amenorrhea.[3] Excessive drowsiness has also been observed. Epileptiform convulsions are a common symptom (Horsley). Binasal hemianopsia may also occur.[4] An excessive development of fat with defective development of the genital organs has been observed in some cases of tumor in this region. (*Adiposis cerebralis* or *dystrophia adiposogenitalis of* Frohlich.) Symp-

[1] Mills, New York Med. Jour., February 11, 1905, p. 266.
[2] Collier, Brain, 1904, p. 490.
[3] Cushing, Jour. Nerv. and Ment. Dis., November, 1906, p. 704.
[4] Shoemaker, New York Med. Jour., February 4, 1905.

toms of acromegaly are not always present. Other cranial nerves may be affected by the growth. The skiagram will show enlargement of the sella turcica.

Diagnosis.—The combination of two or more of the general symptoms mentioned with focal symptoms should cause a strong suspicion of the existence of a brain tumor; the diagnosis, however, may at times be difficult; if the growth has undergone calcification the *x*-rays will be of service; the following may be mistaken for it:

Brain abscess produces similar symptoms, but they usually develop after head injuries, or a history of the previous existence of suppuration in the middle ear or some of the other causes mentioned in the etiology of brain abscess is obtainable.

The symptoms develop more rapidly and with greater severity than those of tumor; leukocytosis will be present, and there may be fever with slow pulse. Headache is more severe and papilledema is more apt to be present in tumor. In abscess, after the appearance of symptoms, they may disappear for some time; a duration of one to two years, in which symptoms are constantly present, indicates tumor.

Tuberculous meningitis, in which the symptoms pursue a chronic course, may be mistaken for tumor. When meningitis is present, the headache is usually more severe and diffuse; hyperesthesia of the skin and special senses is more apt to be present. Optic neuritis is not so frequent, and when present is not so intense, and tubercles upon the choroid may be present. Irregular but persistent fever would point to meningitis. Examination of fluid obtained by lumbar puncture (pp. 96 and 227) will usually assist. It must be remembered that the two may coexist, in which case the diagnosis might be impossible, as would also be the case when the meningitis is localized to a small area.

Chronic hydrocephalus is often the result of tumor, especially when it is situated in the cerebellum. In those cases which are due to inflammation of the ependyma, the general symptoms are similar to those of tumor, but the local symptoms are somewhat different. Spastic paralysis develops without spasm and is always bilateral. The course is

very chronic and atrophy of the cranial bones occurs; optic neuritis is not so common (p. 230).

Apoplectic attacks, which sometimes occur in tumor, are distinguished from the ordinary cerebral hemorrhage by the discovery of the symptoms of tumor after those of the hemorrhage have subsided.

Uremia may produce symptoms very similar to those of tumor; even choked disk is sometimes caused by nephritis. The presence of albumin and casts would point to uremia, as would also the sudden development and transient nature of the symptoms; of course, tumor and nephritis may coexist.

Chronic anemia may simulate tumor even to the development of changes in the eye-ground resembling those of tumor; a history of long-standing anemia, the characteristic changes in the number, etc., of the corpuscles, and the relief of the symptoms by appropriate treatment would distinguish the two.

Symptoms resembling those of **neurasthenia** and **hysteria** may occur in the early stages, and in some cases prevail throughout the course of brain tumor. These conditions are especially apt to be confounded in growths situated in the frontal lobes. Careful investigation will usually detect the presence of organic disease, if it exists.

Growths in the frontal lobes may also cause symptoms simulating those of *paresis* (p. 415).

Having concluded that a tumor exists, it is then necessary to decide upon the location and variety of the growth. The means of deciding the former have been detailed under focal symptoms. The latter is often difficult. The peculiarities of the different varieties have been detailed on page 341. The probabilities are tabulated by Dana as follows:

Childhood	Tubercle, parasites.
Early life	Gumma, glioma, parasites.
Early and middle life	Sarcoma, glioma, gumma.
Middle and late life	Sarcoma, gumma, cancer.

Prognosis.—Unless the tumor can be removed or the symptoms alleviated by surgical means the prognosis is

unfavorable. The average duration of the symptoms is three years. Although cases may occur in which the patient lives in comparative comfort for a much longer time. Sudden death may be due to tumor. Blindness, especially in subtentorial growths, may occur early. Cases have been recorded which had all the symptoms of cerebral tumor, but which recovered under medical treatment.[1]

Treatment.—Medical treatment, excepting when the growth is syphilitic, when vigorous antisyphilitic treatment is indicated, is symptomatic. The headache is treated by the measures usually employed for the relief of that symptom. Vomiting and vertigo may be relieved by the use of hydrobromate of hyoscine, gr. $\frac{1}{100}$, every four hours, or by morphine and atropine hypodermically. Convulsions are treated by the usual methods employed for epilepsy.

It is well in all cases to try the effect of specific treatment. This must be vigorous, inunctions of mercury being employed as well as iodide of potassium. The latter drug often for a time relieves the symptoms of non-syphilitic tumors. If syphilitic, there should be marked improvement in the course of a few weeks. The presence of the Wassermann reaction may be, in the presence of corroborating symptoms, of help in establishing the syphilitic origin of a neoplasm. It must be remembered, however, that while the reaction may be due to a previous infection the growth is not necessarily due to that infection, and may be another form of tumor.

Surgical treatment, when the tumor can be localized and reached, should be resorted to if this treatment fails. Tumors situated in or near the cortex are those suitable for operation. Fibroma and sarcoma are the most favorable; but glioma and gliosarcoma have been removed, but are liable to return. About 7 per cent. of tumors are operable.

Tumors involving a lateral lobe of the cerebellum and those of the cerebellopontile angle have also been removed.[2] The latter is a much more difficult operation than the former.

[1] Hoppe, Jour. Nerv. and Ment. Dis., February, 1907, p. 97.
[2] Frazier, New York Med. Jour., February 11, 1905, p. 272.

Life may be prolonged, headache and the occurrence of blindness prevented by operation even when the growth is of such a nature or so situated that it cannot be removed. This consists in relieving pressure by operation now known as "decompression," and consists in opening the skull, the technique of which is described in works on surgery.[1] It should be done early, especially if papilledema is pronounced.

[1] Frazier and Spiller, Univ. Penna. Med. Bull., September, 1906.

CHAPTER XII

FOCAL AND DIFFUSE DISEASES OF THE SPINAL CORD

SPINAL LOCALIZATION

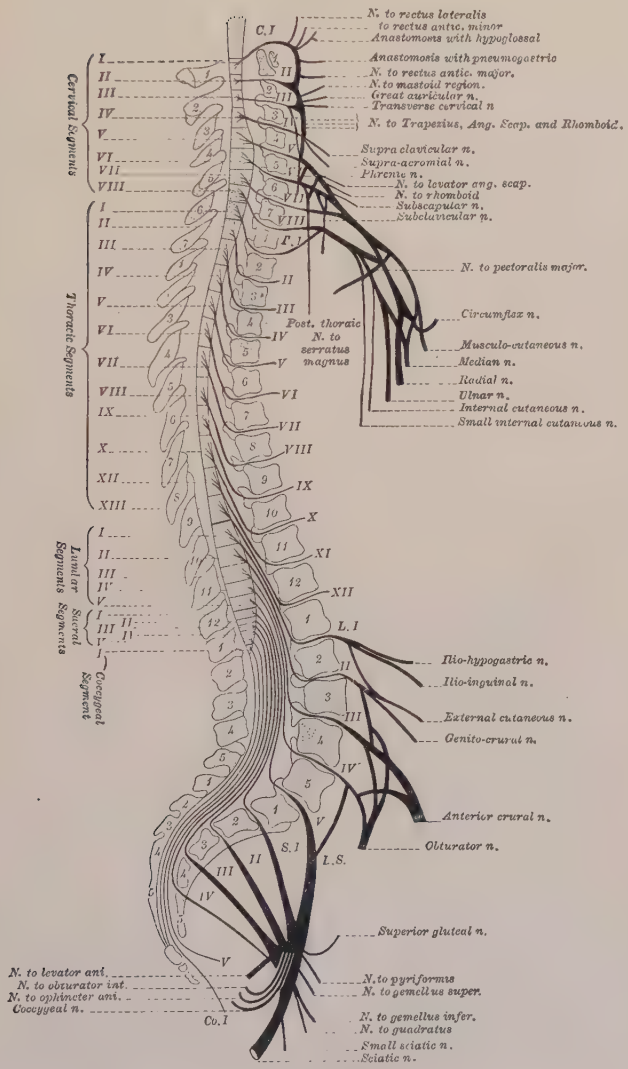
Reflex and Vasomotor Centres.—The spinal cord, in addition to the carrying of motor impulses by means of the pyramidal tracts from the brain to the peripheral nerves, and of sensory impressions from the skin, muscles, and joints to the brain (see Motor and Sensory Tracts), contains in its gray matter a number of cells, some of which are the parent cells of the fiber which form the motor fibers of the peripheral nerves, and which also maintain the nutrition of the muscles and act as reflex centres (Figs. 14 and 17) and vasomotor centres. These are situated in the *anterior horns.*

Sensory and Trophic Centres.—Others, situated in the *posterior horns,* receive the sensory fibers from the peripheral nerves; act as trophic centres for the bones, skin, and joints.

The *intermediolateral tract* (p. 68) is a system of cells extending from the eighth cervical to the lower end of the second lumbar segments and situated along the lateral margin of that portion of the gray matter which is intermediate between the anterior and posterior cornua. It is thought probable that the fibers of the cervical and splanchnic sympathetic nerves arise from these cells and leave the cord by the white rami communicantes. Loss of function of sympathetic nerves (anidrosis, etc.) has been associated with disease of these cells, through them also visceral and vasomotor functions suffer in spinal cord disease.

Spinal Automatic Centres.—In the extreme lower part of the cord, situated about the central canal, are groups of cells known as the spinal automatic centres—*i. e.*, the *genital,*

FIG. 102

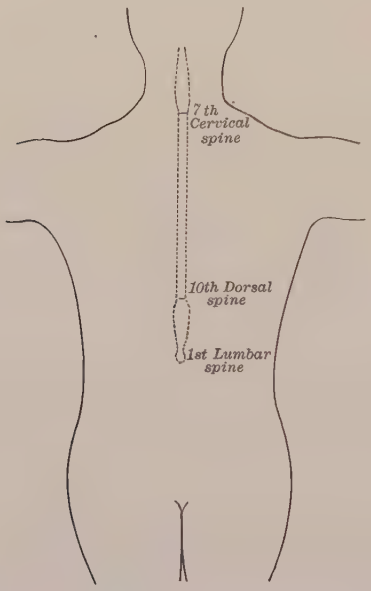


The relations of the segments of the spinal cord and their nerve roots to the bodies and spines of the vertebræ. (Starr.)

presiding over the functions of erection and ejaculation; and the *bladder* and *rectal*, which preside over the functions of urination and defecation. They are controlled as other reflexes are (Fig. 17, p. 60).

Segments.—The spinal cord, therefore, may be looked upon as a series of thirty-one segments placed one above the other, each of which is connected with a pair of spinal nerves,

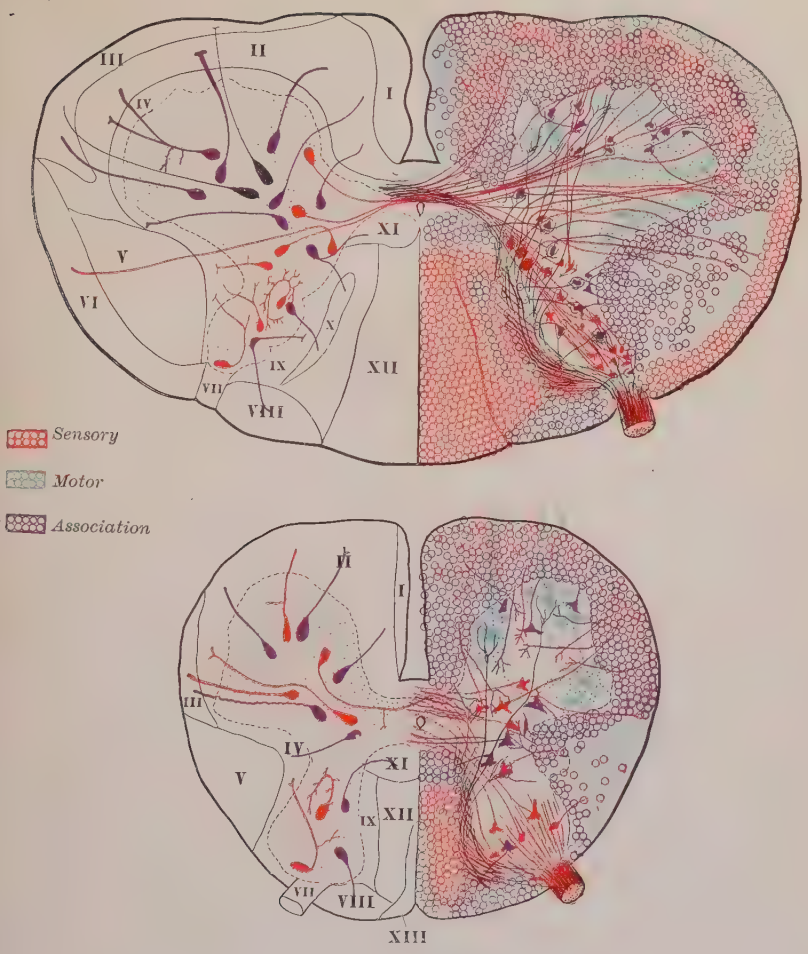
FIG. 103



Relations of the cervical and lumbar enlargements to the vertebræ

contain certain trophic, reflex, and vasomotor centres, and through which run the motor and sensory tracts coming from and to the brain (Plate IV). As the spinal cord only reaches from the foramen magnum to opposite the base of the first lumbar vertebra, it can readily be seen that all of the spinal segments are not directly opposite the vertebra whose name they bear, and that the intraspinal course of the nerves, after they emerge from the cord, is longer the

PLATE IV



The Cervical and Sacral Enlargements of the Spinal Cord in Cross-section. (Starr.)

Showing the various neurons in the gray matter, the direction of their axons, and the varieties of fibers in the different columns of the cord. Blue, motor neurons; red, sensory neurons; purple, association neurons and axons.

I. Ant. median column. II. Anterolateral column. III. Gowers' anterolateral ascending column. IV. Marginal column. V. Lateral pyramidal column. VI. Direct cerebellar column. VII. Lissauer's tract. VIII. Ext. portion of column of Burdach. IX. Root zone of the column of Burdach. X. Descending comma-shaped bundle of Schultze. XI. Post. commissural tract. XII. Column of Goll. XIII. Septomarginal tract.

lower we descend to the cord (Fig. 102). Hence the nerves from the lumbar and sacral segments form a large bundle (the cauda equina), which runs through the spinal canal from their origin in the cord to the vertebræ between which they emerge. The eighth cervical and first dorsal segments lie in front of the spine of the seventh cervical vertebra. The lumbosacral enlargement begins in front of the tip of the spine of the tenth dorsal vertebra (Fig. 103).

Chipault has given the following rules for the determination of the relation of the segments to the spinous processes of the vertebræ: "In the cervical region add one to the number of vertebra and this will give the segment opposite to it. In the upper dorsal region add two; from the sixth to the eleventh dorsal add three. The lower part of the eleventh dorsal spinous process and the space below it are opposite the lower three lumbar segments. The twelfth dorsal spinous process and the space below it are opposite the sacral segments" (Fig. 102).

Enlargements.—The most important parts of the cord are the cervical and the lumbar enlargements.

The former gives origin to the nerves which supply the arms, and contains the ciliospinal, wrist-jerk, elbow-jerk, scapular, palmar, and hypochondrium reflex centres. The latter gives origin to the nerves which go to the legs, and contains the centres for the plantar, gluteal, and cremasteric reflexes, for the knee and Achilles-jerks, and also the automatic reflex centres mentioned above.

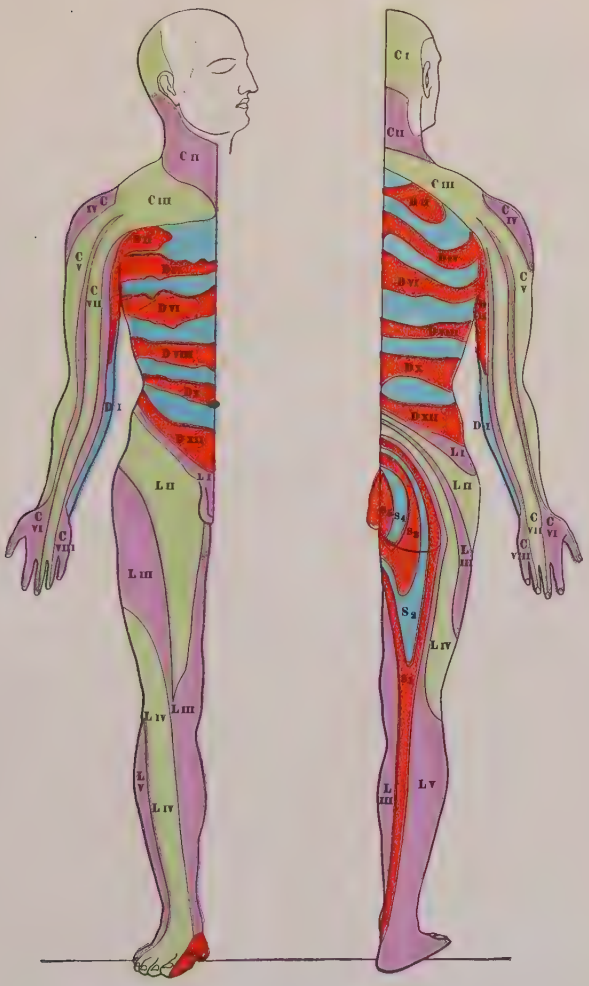
By knowing the particular functions of each spinal segment we are enabled **to locate** the situation and extent of a focal lesion in the cord. It must be remembered that destruction of one segment will not produce complete atrophy and disability of a muscle, as most muscles are supplied from more than one segment, usually three (p. 371). A focal lesion of the cord cannot damage a single muscle alone, but will weaken a number.

The following table, first made by Starr, and elaborated by Mills and others, shows the functions of each segment. It is still, however, far from being perfect, and further modifications may be made in it in the future.

Localization of the Functions of the Segments of the Spinal Cord

Segment.	Muscles.	Reflex.	Sensation.
Second and third cervical.	Sternomastoid. Trapezius. Scaleni and neck. Diaphragm.	*Hypochondrium* (?) (third to fourth cervical). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of neck and of head to vertex. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coracobrachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	*Pupillary* (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, musculocutaneous, or external cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coracobrachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscle of shoulder blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	*Scapular* (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of scapular muscles. Biceps, tapping biceps tendon causes contraction of that muscle. *Supinator longus* (fourth to fifth cervical). Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to wrist. (Supraclavicular, circumflex, musculocutaneous, or external cutaneous, radial.)
Sixth cervical.	Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part.) Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	*Triceps* (sixth to seventh cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (sixth to eighth cervical). Tapping tendons causes extension of hand.	Outer side and front of forearm. Back of hand, radial distributions. (Chiefly musculocutaneous or external cutaneous,internal cutaneous.)
Seventh cervical.	Triceps (long head). Extensors of wrists and fingers. Pronators of wrist. Flexors or wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	*Anterior wrist* (seventh to eighth cervical). Tapping anterior tendon causes flexion of hand. *Palmar* (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index and one-half middle finger. (Musculocutaneous or external cutaneous, internal cutaneous, radial median.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand muscles.	. . . . .	Ulnar area of hand, back, and palm, inner border of forearm (internal cutaneous,ulnar)
First dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	. . . . .	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)

PLATE V



Areas of Anesthesia upon the Body after Lesions in the Various Segments of the Spinal Cord. (Starr.)

The segments of the cord are numbered: C I to VIII, D I to XII, L I to V, S 1 to 5, and these numbers are placed on the region of the skin supplied by the sensory nerves of the corresponding segment.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD—CONTINUED

Segment.	Muscles.	Reflex.	Sensation.
Second dorsal.	. . . . .	. . . . .	Inner side of arm near and in axilla. (Intercostohumeral.)
Second to twelfth dorsal.	Muscles of back and abdomen. Erectore spinæ.	*Epigastric* (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium.	Skin of chest and abdomen, in bands running around and downward and corresponding to spinal nerves, testicle, eleventh and twelfth dorsal.
		Abdominal (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly.	Upper gluteal region. (Intercostals and dorsal posterior nerves.)
First lumbar.	Iliopsoas. Rectus. Sartorius.	*Cremasteric* (first to third lumbar). Stroking inner thigh causes retraction of testicle.	Skin over groin, testicle, and front of scrotum. Iliohypogastric, ilioinguinal.)
Second lumbar.	Iliopsoas. Sartorius. Quadriceps femoris.	. . . . .	Outer side of thigh. (Genitocrural, external cutaneous.)
Third lumbar.	Quadriceps femoris. Anterior part of biceps. Inward rotators ofthigh. Abductors of thigh.	*Patellar* (third to fourth lumbar). Striking patellar tendon causes extension of leg.	Front of thigh. (Middle cutaneous, internal cutaneous, long saphenous, obturator.)
Fourth lumbar.	Abductors of thigh. Adductors of thigh. Flexors of knee. Tibialis anticus. Peroneus longus.	*Gluteal* (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)
Fifth lumbar.	Outward rotators of thigh. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	*Achilles tendon* (fifth lumbar to first sacral). Overextension causes rapid flexion of ankle, called ankle clonus. Also centre for the Achilles-jerk.	Back and outer side of leg; sole; dorsum of foot. (External popliteal, external saphenous, musculocutaneous plantar.)
First and second sacral.	Flexors of ankle. Long flexor of toes. Intrinsic foot muscles.	*Plantar* (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back and outer side of leg; sole; dorsum of foot. Same as fifth lumbar.)
Third, fourth, and fifth sacral.	Gluteus maximus. Perineal. Muscles of bladder, rectum, and external genitals.	Vesical centres. Anal centres.	Back of thigh, anus, perineum, external genitals. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)
Fifth sacral and coccygeal.	Coccygeus muscles.	. . . . .	Skin about the anus and coccyx. (Coccygeal.)

The **areas of anesthesia** caused by lesions of the segments are shown in Plate V and Figs. 104 and 105. As the diagrams

made by different authorities differ somewhat, two are given. If the pathways in the cord which conduct sensations are injured by the lesion, the sensory impressions received by the segments below the seat of the lesion would be prevented

Fig. 104

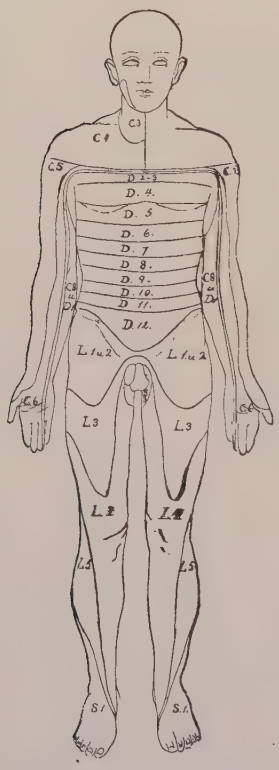
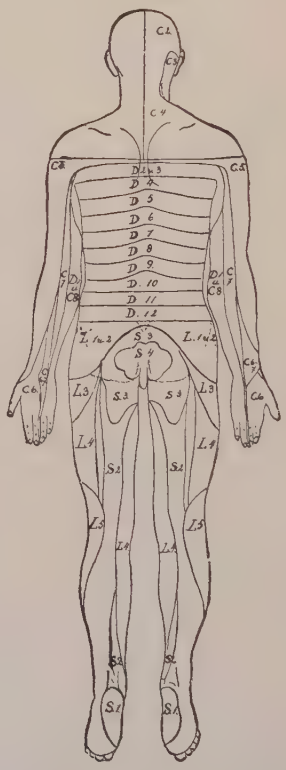


Fig. 105



Showing the regions innervated by the different spinal roots or the corresponding segments of the cord. It should be remembered that the limits are not in reality so sharply defined, but extend into one another. (Kocher.)

from reaching the cerebrum, and hence there would likewise be anesthesia in the areas supplied by these segments. For instance, if a lesion destroyed the cord at the ninth dorsal segment, in addition to the sensory area represented in that

segment, there would be anesthesia in the areas represented in all the segments below the first, which would involve both legs and the body to the umbilicus (Figs. 104 and 105). On the other hand, if the lesion is confined to the nerve roots, the sensory tracts of the cord escaping, the area of anesthesia is confined to the area represented by that particular segment or segments (segmental, spinal, or root type of anesthesia). This type is frequently seen in tabes, and in cases of spinal caries when the nerve roots only, and not the cord itself, are involved, and also in tumor. It has, moreover, been shown by Sherrington that the areas supplied by the different spinal roots overlap, and that hence disease affecting one

FIG. 106

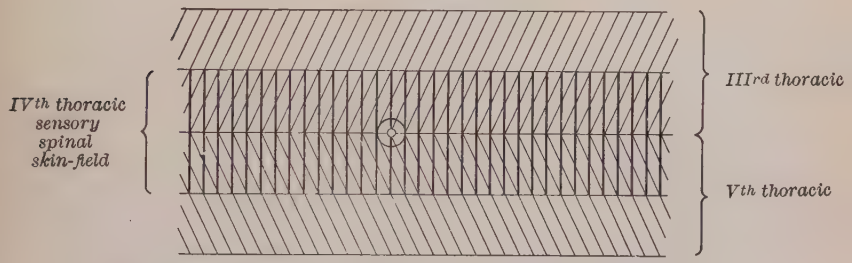


Diagram showing the nerve supply of the skin of the thorax; every part is supplied by two nerves. (Sherrington.)

root only would not cause anesthesia, as its area is supplied from the next roots both above and below. Therefore, to have anesthesia of this type present, the root next above and that next below that segment to which the area of anesthesia refers must also be involved (Fig. 106). Anesthesia due to peripheral-nerve lesions is confined to the anatomical distribution of the nerves in the skin (Figs. 47 and 48).

For instance, if there was complete paralysis of both legs, with atrophy of the flexors and adductors of the thigh and extensors of the leg, loss of the cremasteric and patellar reflexes, but with preservation of ankle clonus and of the plantar reflex, together with incontinence of urine and anes-

thesia involving both legs and extending upward to a line a little above the symphysis pubis, we would know that there was a transverse lesion probably extending from the first to the third lumbar segment. In such a case, while there would be complete paralysis of both legs, atrophy would only be present in the muscles mentioned, because the cells which exercise trophic influence over them are situated in the cord from the first to the third lumbar segments inclusive. Below the level of the third lumbar segment the cells are intact and exercise their functions; but the muscles are not capable of voluntary movement, because impulses from the brain are stopped at the lesion; likewise we have anesthesia because sensory impulses cannot get up. Often at the level of the lesion there is a zone of hyperesthesia, due to irritation of the posterior nerve roots situated in this region.

A **zone of anesthesia** extending to the umbilicus corresponds to the ninth dorsal segment; one inch below this, to the tenth[1] and the nipples to the fourth dorsal. Loss of sensibility in the arms is not present in lesions below the first dorsal.

A **lesion that damages the fifth cervical segment** and spares those below causes a characteristic group of symptoms. The fifth segment supplies chiefly the biceps, brachialis anticus, deltoid, and supinators. Hence if these muscles are paralyzed the elbow will lie next the body, and the forearm and hand will lie prone on the chest (Fig. 107). If, however, the fifth segment escapes, the lesion extending even as high as the sixth, the elbow will be abducted by the deltoid, the forearm flexed by the biceps, the supinators will supinate the forearm, and the infraspinatus will rotate the humerus externally, and the patient will assume the position shown in Fig. 108.

In lesions about the *tenth dorsal segment*, when the patient attempts to raise the shoulders from the bed, the umbilicus will be noticed to move upward a half inch or more. This is due to the fact that a lesion in this location causes

[1] Spiller and Weisenburg, Review of Neurol. and Psychiatry, October, 1904.

paralysis of the recti muscles below the umbilicus only, therefore in the attempt to raise the shoulders the recti above the umbilicus will contract and pull it upward.

Fig. 107

An attitude in a case in which the fifth cervical nerve had been injured on the left side only. (Thorburn.)

Fig. 108

Peculiar attitude of a patient in whom the fifth cervical nerves had not been crushed. (Thorburn.)

Lesions located in that part of the cord extending between the fourth and fifth lumbar, as the upper, and the second and third sacral segments, as the lower limit, termed by Minor the **epiconus medullaris,**[1] give a characteristic group

[1] Progressive Medicine, 1902, p. 265.

of symptoms, consisting of present knee-jerks, absent Achilles-jerks, intact sphincters, sensory paralysis limited to the distribution of these segments (Figs. 104 and 105), and motor paralysis, especially marked in the peroneal muscles, with atrophy and De R, producing a steppage gait. Other muscles of the leg may also be affected.

Fig. 109

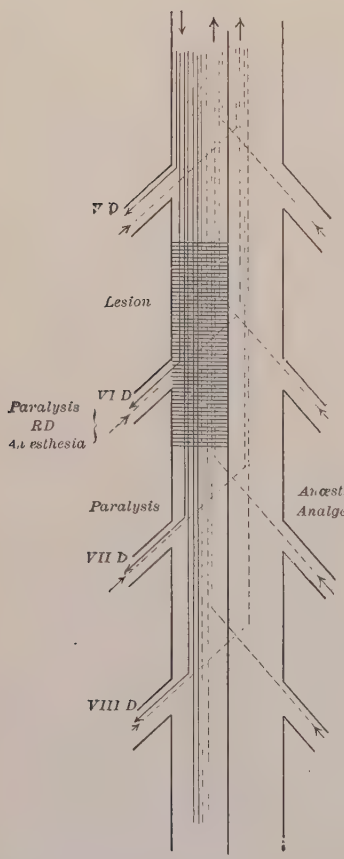


Diagram showing the effect of a unilateral lesion of the spinal cord, causing "Brown-Séquard paralysis." (Starr.)

Lesions of the *conus medullaris*, which includes that part of the cord extending from the filum terminale to and including the third sacral segment are characterized by absence of paralysis of the limbs and by paralysis of the bladder and rectum, there being incontinence due to relaxed sphincters; loss of sexual power, and a saddle-shaped area of anesthesia involving the skin about the anus, perineum, scrotum, penis, mucous membrane of urethra and anus; the testicle is sensitive, its nerve supply originating higher up (p. 369) (Figs. 102, 104, and 105). Lesions of the *cauda equina* often simulate conus lesions, producing the same area of anesthesia and sphincter paralysis; they are, however, apt to be associated with radiating pains in the course of the

nerve trunks, and if the lesion is extensive, progressive paralysis of both motor and sensory nerves supplying muscles and skin of the legs occurs. The symptoms also are apt to be not so symmetrical as in conus lesions. If the lesion is a fracture of a vertebra below the first lumbar the cauda is involved. If a hemorrhage the blood is apt to gravitate downward, and principally affects the nerve roots from the lower sacral segments, and such cases are especially apt to simulate conus lesions (Fig. 102). (See pp. 204 and 397.)

In **complete transverse lesions**, those in which the cord at the seat of the lesion is completely destroyed, the deep reflexes behave differently from the way they do in partial lesions. In the latter those controlled by centres below the seat of the lesion are increased, but in the former case they are **permanently abolished.**[1]

Hemilesions of the cord produce a characteristic group of symptoms, known as Brown-Séquard paralysis. In typical cases these are distributed according to the following table slightly modified from Gowers:

CORD

		Lesion.	
Level of the lesion.	Zone of cutaneous hyperesthesia.		
	Zone of cutaneous anesthesia.		
	Paralysis with atrophy of muscle supplied by the segments involved.		
Below the lesion.	Paralysis without atrophy below seat of lesion.		Muscular power normal.
	Hyperesthesia of skin.		Loss of pain and temperature sense; tactile sense impaired sometimes.
	Muscular sense impaired.		Muscular sense normal.
	Reflex action, first lessened, then increased.		Reflex action normal.
	Temperature raised.		Temperature same as that above the lesion.

The *sensory symptoms* are peculiar. In the early stages there may be some loss of tactile sensibility on the side of the lesion, which soon disappears. On the side opposite the

[1] In acute partial transverse lesions they may be temporarily abolished, due to irritation of the inhibitory fibers, but return and usually become increased.

lesion all forms of sensation, except muscular, may be impaired; but, as a rule, it is only pain sense and temperature sense that are lost; this is due to the early decussation of their fibers (p. 27) (Fig. 109).

SPINAL BLOODVESSELS

The main arteries of the spinal cord are three in number. They are situated on the anterior and posterior surfaces of the cord, along its entire length. They arise from the vertebral arteries, the anterior spinal artery passes down the anterior surface of the cord in front of the anterior median fissure. The two posterior spinal arteries pass downward on each side near the point of entrance of the posterior nerve roots. There are also a number of smaller vessels, branches of the intercostal arteries, which are distributed to the anterolateral surfaces of the cord. They freely anastomose with each other (Fig. 110). They are termed the anterior and posterior radicular arteries. They also anastomose with branches of the anterior and posterior spinal arteries. Those branches which enter the cord are end arteries, consequently obstruction to the circulation in one of them, or its rupture, causes softening in the area supplied by it. Branches from the anterior spinal artery supply most of the gray matter; the white matter is supplied by both the anterior and posterior vessels. The veins originate in plexuses, and pass from the cord with the spinal nerves to reach the vena cava.

AFFECTIONS OF THE BLOODVESSELS

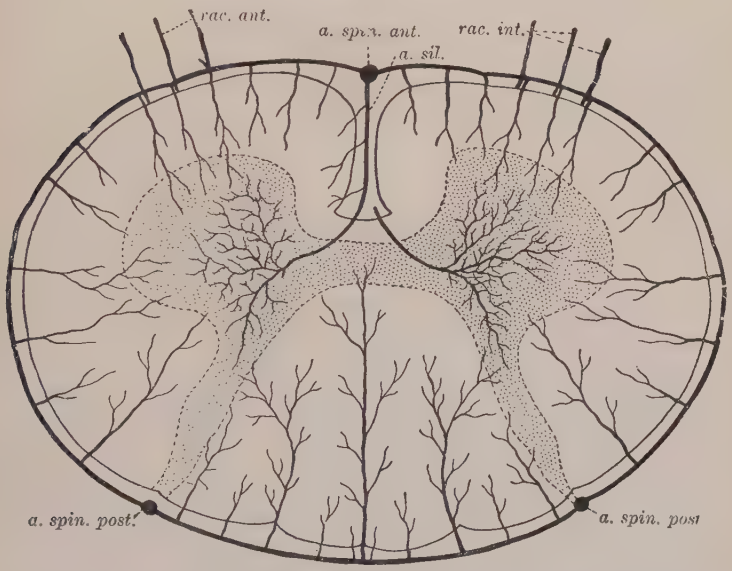
Embolism and thrombosis may occur in the spinal arteries (p. 381). Hemorrhage either into the meninges or the cord substance also occurs.

Spinal Meningeal Hemorrhage (Hematorrhachis).—Etiology.—It occurs in newly born children and adults, and may be inside or outside the dura. The usual causes in the latter are injuries, with or without fracture of the spine. Severe convulsions from any cause, severe muscular exertion,

purpura, and alterations in the blood following infectious fevers, and the bursting of an aortic or vertebral aneurysm may also be causes.

Symptoms.—Small hemorrhages may produce no symptoms. In those large enough to cause them there is sudden severe pain in the back, which extends into the limbs, and

FIG. 110



Scheme to show the course and distribution of the terminal branches of the arterial plexus of the pia mater: *a. spin. post.*, posterior spinal arteries; *a. spin. ant.*, anterior spinal arteries; *a. sil.*, anterior median fissure; *rac. ant.*, anterior root arteries. (After Van Gehuchten.)

is associated with tingling, hyperesthesia, and muscular twitchings (irritation of the nerve roots). Blood may be found in the fluid obtained by lumbar puncture. Later, weakness or paralysis of the limbs, more or less pronounced anesthesia (root type), and disorders of the sphincters occur. The symptoms rapidly reach their height, then subside with either recovery gradually occurring or symptoms of chronic

meningitis remaining. More rarely the blood gravitates to the end of the cord, causing, as the blood gradually fills up the vertebral cavity, symptoms of an ascending paralysis.

Diagnosis.—The diagnosis is based upon the sudden onset of the symptoms following injury or other of the causes mentioned; the presence of severe pain and nerve-root irritation, and the subsidence of the symptoms.

In hemorrhage **into the cord substance** there are less pain and irritation, and more pronounced and permanent motor and sensory paralysis. The spinal fluid will not contain blood (p. 96). When the cord is crushed, due to dislocation or fracture, the symptoms more resemble those of hematomyelia, and external evidences of the injury can usually be found.

Prognosis.—The prognosis is grave, but if death does not occur in a few days the prospect of partial or nearly complete recovery is good.

Treatment.—This consists of absolute rest, the patient being as much as possible on his abdomen and face, cold to the spine, and the administration of remedies to relieve pain and move the bowels. For the later symptoms iodide of potassium, mercury, and counterirritation to the spine may be employed. In cases associated with fracture or dislocation, surgical measures may be indicated (p. 392).

Hemorrhage into the Substance of the Cord (Hematomyelia).—Etiology.—Overexertion, excessive sexual intercourse, exposure, syphilitic or other disease of the blood-vessels, purpura, and convulsions are rare causes. It may occur secondarily in myelitis and spinal tumor. Trauma, as a blow or fall upon the back, is the most common cause and may or may not be associated with fracture of the vertebra. Only the latter is here considered; the former is discussed on page 391. It sometimes occurs in infancy, but is most common between the ages of twenty and forty.

Morbid Anatomy.—The lower cervical region is the most common seat of hemorrhage, which is chiefly in the gray matter. The clot may be absorbed, leaving a cavity, which

may cause the symptoms of syringomyelia. While the hemorrhage is commonly single, multiple hemorrhages may occur.

Symptoms.—These appear suddenly; a feeling of numbness and weakness in the extremities being first noticed, which in an hour or so develops into complete paralysis below the seat of the lesion, with anesthesia and paralysis of the sphincters. The reflexes below the seat of the lesion are at first absent, but soon appear and become exaggerated. There may be some pain in the back. The location of the hemorrhage can be determined by the methods detailed under Spinal Localization. Symptoms of acute myelitis may soon appear and death result, or they may subside in the course of a week or so, leaving a permanent condition similar to chronic myelitis. As the hemorrhage is usually into the gray matter, a symptom-complex resembling syringomyelia often results.

Diagnosis.—The diagnosis is based upon the sudden onset, without prodromes, and absence of fever. The features which distinguish it from spinal meningeal hemorrhage have been mentioned under that condition. It may be mistaken for acute myelitis, which is often produced by it, and is sometimes its cause. It is also often necessary if trauma has occurred to determine if the vertebræ have been injured (p. 391).

Prognosis.—The prognosis is serious; death often occurs, and permanent disability of more or less extent is always the result. Hemorrhages below the cervical region are the most favorable.

Treatment.—Absolute rest, ice-bags to the spine, and remedies to lessen the heart's action. Treatment after the acute attack is the same as that employed in myelitis.

Caisson Disease (Divers' Paralysis). — Definition and Etiology.—By this we mean a more or less complete paraplegia which occurs in persons who suddenly return from a condensed air to the normal atmosphere.

It occurs in those who work in caissons where the pressure is over one atmosphere, and in divers. It rarely occurs when the pressure is not over that; and, also, if the person

has not been subjected to the pressure for at least an hour. Alcoholism, old age, and obesity are predisposing causes.

Symptoms.—Soon (usually within an hour) after the patient comes to the surface he is seized with violent pains in the limbs and sometimes the abdomen, termed "the bends." A difficulty in breathing and cough may also occur, which has been termed "the chokes." If proper precautions are taken nothing further may result. If not, or if the primary attack is more severe, dizziness and staggering occur. There is vomiting, and the pulse is rapid and weak. Purplish spots appear on the body and limbs; dimness of vision and hearing may also be complained of. This form has been termed "the staggers." In some cases symptoms resembling those of myelitis appear (p. 381). In others coma results with complete relaxation, weak, running pulse, normal or elevated temperature, etc.

Morbid Anatomy and Pathology.—Congestion and softening of the cord are found. Microscopically, destruction of the nerve tissue of the posterior and lateral columns in the dorsal region has been found in a number of cases. The pathology of these changes is obscure. The pressure in some cases probably causes overdistention of bloodvessels by paralysis of their walls. When the pressure is removed they are unable to accommodate themselves to the changed conditions, and stasis, congestion, and hemorrhages result. Other cases seem to be due to the liberation of gas emboli, chiefly nitrogen, in the blood and tissues, which block the spinal-capillaries and produce softening.

Prognosis and Treatment.—Most cases recover; but either death may occur, especially in those cases which become comatose, or a permanent weakness or paralysis result, resembling chronic myelitis (p. 387).

Those who work in caissons should ascend into the normal air pressure very gradually. If symptoms appear he should be at once returned into compressed air, at the same pressure he was working in; the pressure is then gradually reduced at the rate of about one-half pound a minute until normal conditions are reached. If the symptoms return, the same thing must be repeated. Stimulating liniments, rapidly interrupted faradic currents, and heat are all useful to relieve

the pain. Phenacetin and similar drugs may be given if the heart is not weak. When it is, cardiac stimulants, as strychnine, caffeine, and adrenalin are useful. In the comatose cases, enemas of hot coffee should be employed, with artificial respiration if necessary. If paralysis results, the treatment is that indicated for chronic myelitis[1] (p. 389).

INFLAMMATION OF THE SPINAL CORD (MYELITIS)

This may be acute, subacute, or chronic. If the gray matter only is affected, it is termed *poliomyelitis* (p. 257); if a small vertical extent of both gray and white matter, *transverse myelitis;* if an extensive area of both white and gray matter, *diffuse myelitis;* if a large area of gray matter, *central myelitis;* if in scattered areas, *disseminated myelitis.* According to the cause, we have *hemorrhagic myelitis* when due to hemorrhage; *compression myelitis* when due to vertebral disease or tumor; and tuberculous and syphilitic myelitis.

Acute Myelitis.—Etiology.—Most cases occur between the ages of ten and forty years. The causes are injuries causing laceration or bruising of the cord, or hemorrhage within its substance. There may be either fracture or dislocation of the vertebræ, or blows or falls upon the back without apparent bone injury, the symptoms in such cases may not develop until a number of days after the injury;[2] hemorrhage occurring independently of trauma; extension of inflammation from neighboring organs, as spinal meningitis, the inflammation extending from the membranes to the cord; exposure to cold and wet; toxic agents—*i. e.,* infectious diseases, syphilis, and more rarely the metallic poisons. When due to metallic poisoning, multiple neuritis usually coexists. In many cases no cause can positively be determined. Symptoms resembling myelitis so closely that they can rarely be distinguished are caused by *thrombosis* or *embolism* in the vessels of the cord with consequent softening. It is termed *myelomalacia,* and may be discussed with myelitis.

Morbid Anatomy.—This presents three stages. In the first the membranes of the cord are usually more or less

[1] Pelton, Amer. Jour. Med. Sci., May, 1907, p. 679.

[2] Warrington, Rev. Neurol. and Psychiat., May, 1910, p. 271.

opaque. The substance of the cord is softer than normal, and may be either swollen or shrunken. Upon section the surface is pink, and there may be minute hemorrhages (red softening). *Microscopically*, there is great congestion of the bloodvessels, more marked in the veins; a considerable amount of perivascular cellular exudate, the majority of the cells being polynuclear leukocytes. The myelin sheaths show evidences of degeneration; the axis cylinders are swollen and granular, the neuroglia cells are sometimes swollen and increased in number, and the neurogliar tissue is looser than normal. The ganglion cells also show evidences of degeneration. If the process has lasted any time, there is a considerable accumulation of granular cells.

In the next stage section of the cord shows the color to be yellow (yellow softening). The bloodvessels are shrunken, the cellular exudate is still present, but the cells show evidences of fatty degeneration. Compound granular cells are abundant. The nerve fibers and ganglion cells are degenerated. If a severe case, the substance of the cord may consist of a puriform mass consisting of fat globules, granular cells, and detritus.

In the third stage, that of gray degeneration, there is secondary distention of the bloodvessels, causing the cord to assume a redder appearance than is found in the previous stage, and proliferation of connective tissue. Granular cells are principally found in the neighborhood of the bloodvessels. The nerve fibers and ganglion cells have mostly disappeared, and secondary degenerations are found above and below the seat of the lesion. In *myelomalacia* the softening may be either red, if accompanied by minute hemorrhages and transudation of blood; white, if the condition has been one of simple death, or yellow if fatty degeneration has taken place. There is a hyperplasia of the neuroglia, with nuclear formation, and if the patient survives, the products of necrosis are absorbed and fibrous tissue takes their place. The appearances may closely resemble those of myelitis.

Symptoms.—The most common type is *acute transverse myelitis*. The first symptom noticed is a feeling of numbness in the feet and legs. Weakness soon develops, and in the course of a day or so, sooner in some cases, a complete

bilateral paralysis of motion and sensation has developed. The location of the paralysis depends upon the location of the lesion, determined by the methods detailed under Spinal Localization. The usual location is the dorsal or lumbar region, when a paralysis of both legs results. Either retention or incontinence of urine may be an early symptom There may be some fever.

After the symptoms have fully developed there will be found more or less complete paralysis of the legs, loss of tactile, pain, temperature, and muscle sense in various degrees, extending up to nearly the lowest limit of the lesion.[1] If the seat of the lesion is below the dorsal region the sensory lesion will not involve all of the legs, but its extent will depend on the particular segments involved (Figs. 104 and 105). At the level of the upper limit of anesthesia there is a feeling as if a tight band were about the body (girdle pain), above which there is often a zone of hyperesthesia. There may also be pains in the extremities due to meningeal and nerve-root irritation. If these are not affected, the legs feel numb and heavy, but there is neither pain nor tenderness. The sphincters of the bladder and bowels are affected according to the location of the lesion. If dorsal, the bladder is anesthetic, and the urine retained, and at first may be voided at intervals involuntarily (p. 65). In time, if it is not drawn, an incontinence from overflow will develop. The rectum is also anesthetic and the bowels constipated; when they do move the patient is unaware of it. In lower lumbar and sacral lesions sexual power is abolished; in those situated above this region priapism may occur. The legs after a few days feel cold. Bed-sores, oftenest upon the buttocks and heels, develop early. Muscles whose nerve supply originates in the diseased area atrophy, exhibit reactions of degeneration, and the reflexes of that region are absent. Those *below* the seat of lesion become spastic, and contractures develop and the reflexes are increased.[2] (See Spinal Localization.)

[1] The nerves emerge at a lower level than their origin in the cord. (See Segments of Cord, p. 366.)

[2] As has been before stated, in lesions which completely divide the cord the deep reflexes are lost.

When the cervical region is attacked the arms are also paralyzed; unequal or dilated pupils from involvement of the ciliospinal centre and sometimes optic neuritis occur.

If the patient lives a few weeks, some improvement sets in. Sensation first begins to return, then motion; so that in from a few months to a year, walking again becomes possible. but as a secondary degeneration in the motor and sensory tracts has occurred, the patient is left with symptoms resembling those of ataxic paraplegia (p. 287).

The myelitis may, from the original focus, extend either up or down the cord (diffuse myelitis), causing a progression of the symptoms accordingly. If it extends up, Landry's syndrome occurs (p. 271).

The accompanying table shows the principal symptoms present in lumbar, dorsal, and cervical myelitis (Prince):

	Lumbar myelitis.	Dorsal myelitis.	Cervical myelitis.
Paralysis.	Paraplegia.	1. Dorsal, abdominal, and intercostal muscles according to height of lesion. 2. Legs.	Neck muscles, diaphragm, arms, trunk, and legs.
Sensation.	Pains in legs, or girdle pains around loins; hyperesthetic zone around loins; anesthesia of legs, complete or uneven distribution.	Girdle pain and hyperesthetic zone between ensiform cartilage and pubes.	Hyperesthesia and pains in certain nerve distributions of arms; below this, anesthesia of arms, body, and legs.
Atrophy.	Of legs.	Of dorsal and abdominal (and intercostal muscles, not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs.	Atrophy of neck muscles (rare) or more commonly of arms.
Electrical reaction.	De R in atrophied muscles or in mild cases quantitative diminution.	De R in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted.	De R in atrophied muscles.
Bladder.	Incontinence from paralysis of sphincter.	Retention, or intermittent incontinence from reflex actions; later from overflow. Cystitis common.	Same as in dorsal myelitis.
Bowels.	Incontinence from paralysis of sphincter, disguised by constipation.	Involuntary evacuation from reflex spasm, or constipation.	Same as in dorsal myelitis.
Reflexes, superficial.	Lost.	Temporary loss, then rapid increase.	Same as in dorsal myelitis.
Reflexes, deep.	Lost.	Temporary loss, then slow increase.	Same as in dorsal myelitis.
Priapism.	Absent.	Often present.	Often present.

Disseminated Myelitis.—The symptoms of this form usually follow either an infectious disease or septic process. Encephalitis frequently coexists (p. 336). The symptoms resemble those of multiple sclerosis, from which it may be difficult to distinguish. Fever and a very acute onset are characteristic of myelitis.

Diagnosis.—Difficulty should only occur in mild or partial cases. The diseases with which transverse myelitis may be confounded are central myelitis, poliomyelitis, disseminated myelitis, hemorrhage, embolic or thrombotic softening (myelomalacia), multiple neuritis, lesions of the cauda equina, Landry's disease, spinal meningitis, and hysterical paralysis. From *central myelitis* the diagnosis is difficult. These cases are very malignant; and as the symptoms are due to an inflammation of the gray matter which extends up and down the cord for a considerable distance, they are diffuse; there being considerable fever, and, owing to the extensive damage to the trophic centres of the muscles, widespread atrophy. If, as rarely occurs, the central myelitis is focal, atrophy is limited to certain groups of muscles, and dissociation of sensation may be present. These cases resemble syringomyelia, and are usually due to trauma.

In *poliomyelitis* sensation is not affected, and there is no involvement of the sphincters; bed-sores and girdle pain do not occur. The onset of *hemorrhage* is usually more sudden than that of myelitis. The fact that myelitis may occur secondarily to hemorrhage must be borne in mind.

In *disseminated myelitis* the symptoms are scattered, and appear as paralysis of separated groups of muscles and patches of anesthesia irregularly distributed.

As has been said, *myelomalacia* usually cannot be distinguished. If the symptoms develop in one liable to have vascular degeneration and other causes can be excluded it is probably the cause of the trouble. Syphilitic cases are usually so caused.

Multiple neuritis differs in the absence of sphincter involvement, girdle pain, bed-sores, extensive areas of anesthesia, and the less rapid development of the paralysis, while pain and tenderness over the nerve trunks are present.

Lesions of the cauda equina simulate usually those of the lower part of the cord (pp. 204 and 374). The symptoms, however, are less likely to be symmetrical than those of the conus, and some pain in the course of the involved nerves is present. Bed-sores do not usually occur.

In *Landry's* paralysis there is absence of sensory disturbances, sphincter paralysis, muscular wasting, and atrophy (p. 336).

In *meningitis*, hyperesthesia, pain, and muscular spasms are present; paralysis is not so marked, the sphincters escape, and bed-sores do not occur. More or less meningitis is often associated with myelitis. *Hysterical paraplegia* is distinguished by the absence of atrophy, trophic changes, normal movement of toes when sole is irritated (see Babinski Reflex, p. 61), incontinence of urine and feces and the presence of hysterical stigmata.

Prognosis.—The prognosis is serious. Patients who do not die rarely recover full power. Myelitis involving the cervical region is most dangerous to life. High temperature is a bad sign. The milder the degree of paralysis the better the prognosis as regards recovery of function. Hope of such result should not be lost until at least a year has elapsed. Patients may remain bedfast for a year or more, and, finally, die of exhaustion due to bed-sores and cystitis.

Treatment.—For the attack, rest in bed in the prone position (a large part of the time) on a water-bed, if possible; leeches or wet cups applied to the spine; a calomel purge; diaphoresis and small doses of aconite are indicated. Salicylates have been thought to do good. The bladder should be carefully watched, hexamethylenamin being administered, the urine drawn at intervals, and frequently examined, great care being taken to insure cleanliness of the catheter. If cystitis develops, appropriate treatment must be instituted. Enemas must be given if constipation is present. After a week small doses of iodide of potassium may be given. After a month, if fever has subsided, strychnine and the use of electricity should be cautiously begun. Bed-sores must be guarded against by frequent change of position, keeping the skin dry and bathing it with astringent and

hardening solutions (alcohol and alum), and if they appear must be treated by the usual surgical methods. Warm baths may help the contractures. Warm douches to the spine may do good. If it is determined that syphilis is the cause by the history and presence of the Wassermann reaction, a vigorous course of antisyphilitic treatment should be instituted.

Chronic Myelitis.—Etiology.—It may be the sequel of a previous acute myelitis or be due to extension from a meningitis; or it may be caused primarily by syphilis, exposure to cold, alcoholism, infectious diseases, falls, or blows upon the spine.

Morbid Anatomy.—The nervous tissues degenerate or have degenerated, as described under Acute Myelitis, while the neuroglia and connective tissue greatly increases in amount. The bloodvessels become thickened and their lumen diminishes. Secondary degenerations are found. The pia may be thickened and adherent to the cord. To the naked eye the cord is shrunken and harder than normal, and the cut surface is grayish in color.

Symptoms.—These develop much slower than do those of acute myelitis, but when fully developed are essentially the same. Years may elapse before this takes place. The patient at first complains of numbness of the legs and notices that he tires easily; from that weakness gradually progresses, but it may be years before he is rendered helpless. Sensory disturbances also appear: there may be some but not severe pain,[1] tinglings and numbness of the legs, and anesthesia, which is usually not so pronounced as in the acute form. As the gray matter is involved, there is atrophy, with reactions of degeneration in the muscles supplied by the diseased segments. Disturbances of the sphincters, varying according to the location of the lesion, develop, inability to hold the urine being the most common (p. 65). Before the entire thickness of the cord is involved the symptoms resemble, according to the part of the cord then affected,

[1] If the meninges are involved (meningomyelitis) there may be severe pain in the limbs and around the body (girdle pain).

some of the *system* diseases. For instance, the gait may be ataxic when the posterior columns are damaged; when the lateral columns are principally affected the symptoms resemble those of lateral sclerosis; if both lateral and posterior columns suffer, ataxic paraplegia occurs. After the entire thickness of the cord is involved the symptoms—motor, sensory, trophic, etc.—resemble those of the acute form. These, as regards the condition of the reflexes, distribution of atrophy, rigidity or flaccidity of the muscles, of course depend upon the location of the lesion, which is determined by the methods laid down in the chapter on Spinal Localization and (p. 287).

In disseminated myelitis, as the foci are scattered throughout the cord, all possible combinations may be present (p. 403).

Diagnosis.—Lateral sclerosis, compression myelitis, and progressive muscular atrophy, amyotrophic lateral sclerosis, chronic meningitis, may be mistaken for chronic myelitis. The existence of sensory symptoms distinguish it from the former.

The atrophy in *progressive muscular atrophy* usually begins in the hand or shoulder muscles, and there are no sensory symptoms and involvement of the sphincters.

Amyotrophic lateral sclerosis would be distinguished by the absence of sensory symptoms and paresis of the sphincters.

Meningitis is characterized by the greater amount of pain, muscular twitchings, hyperesthesia, etc., which are present. Meningitis and myelitis often occur together (meningomyelitis).

It may be confounded with senile paraplegia (p. 389).

The symptoms of compression myelitis are described on page 389.

Prognosis.—The course of the disease usually extends over years. It may cease to progress and remain stationary, which may be either permanent or followed later by an increase of symptoms. All that we can hope to do by treatment is to arrest the progress of the disease. Slight improvement in the symptoms already present may occur if all of the nervous elements are not completely destroyed. Death

may finally occur from exhaustion due to the development of bed-sores and cystitis, or from some intercurrent disease.

Treatment.—The patient must be cautioned against overexertion and physical fatigue. If possible, he should live in a warm, equable climate. If this cannot be done, exposure to inclement weather should be avoided. Daily warm baths are often beneficial.

If syphilis is ascertained to be the cause, antisyphilitic treatment should be given. If this fails, drugs can only do good as tonics and such, especially arsenic and silver nitrate, should be employed. If there is much muscular rigidity, strychnine should be avoided. Good results have been obtained by opening the dura and then closing it (spinal decompression).[1]

The occurrence of cystitis (p. 386) and bed-sores must be guarded against, and if they occur, treated by the usual methods.

The nutrition of the muscles may be kept up by electricity and massage.

Senile Paraplegia.—In those suffering from vascular degeneration, a progressive weakness of the legs may occur, attended with sensations of numbness, normal or increased knee-jerks, and a peculiar shuffling gait. The diagnosis depends upon the age of the patient, evidence of vascular degeneration, and absence of sensory paralysis. The treatment consists of that of the cause (p. 421).

Compression of the Spinal Cord (Compression Myelitis).—**Etiology.**—This is an interruption of the functions of the cord due to slow and continuous pressure. It may occur at any age. It is caused by caries of the vertebra, either tuberculous or syphilitic, usually the former; newgrowths springing from the bone or membranes; carcinoma and sarcoma involving the vertebræ, being secondary to these growths elsewhere; aneurysm of the abdominal or thoracic aorta, and the development of the echinococcus or other form of cyst in the vertebral canal (p. 392).

Morbid Anatomy.—Meningitis is often associated (p. 214). The cord may appear anemic and thinner than normal at

[1] Bailey and Elsberg, Jour. Amer. Med. Assoc., March 9, 1912, p. 675.

the point of pressure; and if the process has been long-continued is much firmer. *Microscopically*, various stages of degeneration of the nerve elements at the point of pressure and secondary degenerations in the different tracts are found.

Symptoms.—The onset of the symptoms is slow, and characterized at first by the development of symptoms indicating irritation of the nerve roots, as shooting pains in the course of the nerve trunks and muscular twitchings. Symptoms similar to those of chronic myelitis then develop.

If due to vertebral caries, kyphosis can usually be detected; there would be tenderness over the diseased vertebra and great pain produced by movements or jarring of the spinal column.

Diagnosis.—The association of the symptoms with any of the causes given determines the diagnosis. An *x*-ray examination is important if any doubt exists as to the cause.

Prognosis.—The prognosis is fair. In the cases due to vertebral caries, these, if recognized early and subjected to proper treatment, often recover a considerable amount of function. Cases due to either aneurysm or tumor are, of course, hopeless unless in the case of the latter it can be removed.

Treatment.—The treatment of cases due to caries consists of rest in bed, with extension, and the various other mechanical methods employed by surgeons for the treatment of spinal caries, and attention to the bladder and nutrition of the skin and muscles as in myelitis.

If due to syphilis, antisyphilitic treatment should be employed in addition.

In the tuberculous cases cod-liver oil and other tonics, good food, fresh air, etc., are indicated. If this treatment has been employed for a year without improvement, the performance of a *laminectomy* may be considered.[1] It is also indicated if the symptoms appear very acutely, indicating displacement of bone, if marked sepsis is present, or a localized focus of disease is found.[2]

[1] New York and Philadelphia Med. Jour., November 9, 1907, p. 889.

[2] Sachs, Amer. Jour. Med. Sci., December, 1911, p. 815.

INJURIES OF THE SPINAL CORD DUE TO FRACTURED OR DISLOCATED VERTEBRÆ

Etiology.—Such injuries are usually due to either severe falls or blows upon the back or head, as in diving, or to forcible bending.

Morbid Anatomy.—This consists of more or less crushing and disintegration of the cord, with hemorrhages into its substance and into the membranes. It may be divided as with a knife.

Symptoms.—The symptoms depend on the location of the lesion (see Spinal Localization), and consist of loss of function in the parts below the seat of injury. The muscles about the seat of fracture are thrown into spasm and a deformity, usually some kyphosis, can be seen. Motion causes pain at the joint of injury, and in the distribution of the nerves which emerge there. If any doubt exists, a skiagram will show the extent and location of the damage. In the early stages the paralysis is flaccid and the reflexes abolished with incontinence of urine and feces and possibly priapism. A zone of hyperesthesia is above the level of the lesion, below which is complete sensory loss. The Brown-Séquard syndrome may be present (p. 374); if so, it would be evidence that the cord had not been completely destroyed.

Diagnosis.—The important point to be settled is whether the cord has been completely destroyed or not. If any sensation or power of movement exists in parts below the lesion there has not been complete destruction of the cord; this is also the case if the tendon-jerks soon return (inside of a week). If they do not return within this time, provided the lesion does not involve the segments which control the reflex, it is evidence of complete destruction.

Prognosis.—The higher the lesion the worse the prognosis. If the patient survive he is usually left a helpless cripple with incontinence of urine, etc. Now and then, however, great improvement may occur and the patient get about. In lesions of the cauda the prognosis is much better.

Treatment.—At first it consists of rest, etc., as in acute myelitis, and treatment of the shock which exists. After this passes off the question of removing the fragments of bone may be considered. Surgical opinion differs somewhat as to when this should be done if at all. Da Costa[1] says that if the symptoms are significant, we should explore as soon as shock has passed away, even if we think it probable that the cord has been divided; and if it is found divided, it should be sutured. If in any case we are in doubt twelve hours after the injury as to whether or not pressure exists, we should explore. If the case is improving, we should not operate even if there are pressure signs, unless there is a chance that pressure is due to bone, in which case we should operate. Even long after an injury laminectomy may be productive of some benefit. The early stages in these cases are best treated by the surgeon, the function of the neurologist being to localize the lesion. Operation holds out better hope of success when the lesion is so located that the cauda is involved, as the nerves have the power of regeneration, which the cord has not. Operation and removal of compressing fragments of bone, and, possibly, suture of divided roots, or resection and suture of crushed ones, is likely to give good results.

In all these cases, after acute symptoms have subsided, the tone of the muscles should be maintained by electricity, massage, and other measures indicated in chronic myelitis.

TUMORS OF THE SPINAL CORD

Etiology.—With the exception of syphilitic, tuberculous, parasitic, and some cystic formations, we do not know the cause of the formation of tumors. Traumatism may be an infrequent cause,[2] especially of cystic growths. Malignant growths may be secondary to similar growths elsewhere in the body.

[1] Surgery, tenth edition, p. 875.

[2] Bailey, Jour. Nerv. and Ment. Dis., May, 1908, p. 315; Potts, Jour. Nerv. and Ment. Dis., October, 1910, p. 621.

Situation and Varieties.—For clinical purposes they may be conveniently classified into (1) medullary or tumors of the cord; (2) extramedullary or tumors of any of the envelopes, which injure the cord by compression.

Tumors of the first variety are much more rare than those of the second. In 126 cases there were 97 which originated outside the cord, 49 of which were between the dura and the cord, and 19 which developed within the cord, with 14 whose location was doubtful.

Those of the first class comprise gliomata, sarcomata, angiosarcomata, gummata and tubercle.

Those of the second class may be divided into (*a*) vertebral tumors arising from the spinal column or soft tissues immediately surrounding it. They are usually metastatic, either carcinoma or sarcoma. Osteomata, exostoses, chondromata, and myelomata are rare; (*b*) intravertebral tumors which may be divided into two classes, according to their relations with the dura mater.

1. Extradural tumors originating in the periosteum of the vertebra, the outer layer of the dura mater, or the fatty areolar tissue of the epidural space. These are usually either sarcomata, lipomata, fibromata, myxomata, and chondromata. The first two are most common.

2. Intradural tumors originating from either the inner layers of the dura, the arachnoid, the ligamentum denticulatum, the spinal roots or the pia mater.

They may be either diffuse or localized and comprise sarcomata which may or may not be metastatic, endotheliomata, cylindromata, fibromata, lymphangiomata, and cysts (echinococcic or due to other cause). Fibromyxomata and fibrosarcomata are usually found in connection with the nerve roots.

Glioma is always met with within the cord, usually about the central canal, and frequently gives rise to the symptoms of syringomyelia (p. 397), but may be circumscribed.

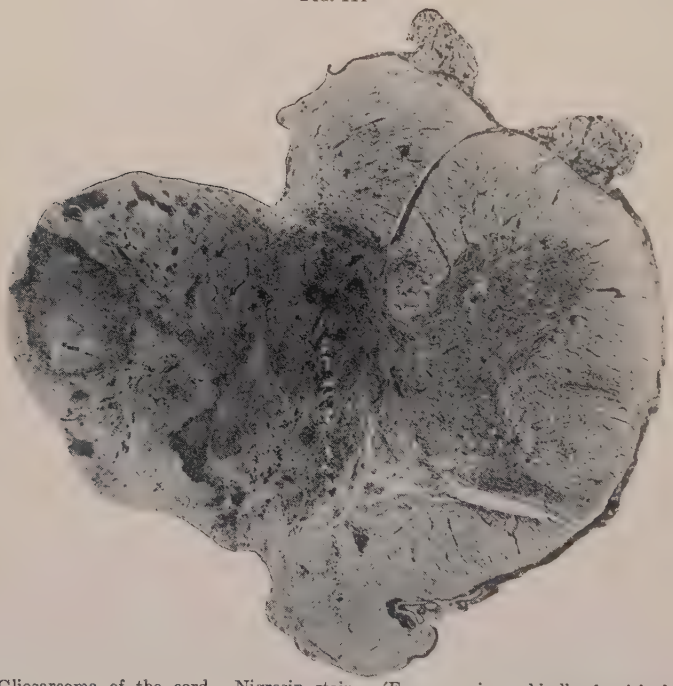
Tumors are usually single, but may be multiple.

Morbid Anatomy.—Tumors developed *outside the cord* compress its substance, producing a compression myelitis

(p. 389). The membrane in the neighborhood of the growth may be edematous and congested.

In tumors of the *cord substance* the cord is enlarged at the seat of growth, and on section the structures of the cord will be observed to be pressed out of their normal position (Fig. 111). The tumor will be firmer than the surrounding

FIG. 111



Gliosarcoma of the cord. Nigrosin stain. (From specimen kindly furnished by E. W. Taylor.)

cord substance, which will be softer than normal. Cavities are frequently formed in gliomatous growths. (See Syringomyelia.) Secondary degenerations occur in the tracts destroyed or pressed upon by the growth.

Symptoms.—*Pain* is usually the first symptom noted. This is due to irritation of the posterior nerve roots, which

produces sharp, shooting pains in the course of the peripheral nerves affected, and to irritation of the sensory tracts of the cord, which causes diffused pain, often occurring in anesthetic areas, which at first are apt to be of the segmental type (Fig. 104). In tumors situated on the anterior surface of the cord, marked pain may be absent.

The anterior nerve roots may be also irritated, producing muscular spasms and rigidity. As the different parts of the cord are either compressed or destroyed, symptoms similar to those of myelitis develop. These may be those of the acute form, but more commonly are those of compression myelitis (p. 389). Ataxia with increased reflexes may be more prominent than paralysis. When limited to one lateral half of the cord, symptoms resembling the Brown-Séquard syndrome develop, which, as the tumor grows, develop into a complete paraplegia. According to Starr, the order in which the symptoms arise is commonly: (1) Peculiar pains of limited distribution; (2) increase of reflexes below the lesion; (3) paraplegia; (4) loss of sensibility; (5) loss of all subjacent reflexes.

Pain while a usual symptom may be absent in some cases.

The course of the disease is usually slow, but tubercle and gumma may develop with great rapidity.

Diagnosis.—This is often uncertain. It is based upon the slow onset and gradual development of the symptoms, associated with symptoms of nerve-root irritation and sensory diminution or loss, especially tactile, in segmental areas (Fig. 104). If malignant growth or tuberculous disease exists, or has existed elsewhere in the body, or there is a history of previous syphilitic infection, the diagnosis is rendered much simpler.

It may be confounded with meningomyelitis, compression myelitis due to vertebral disease, tabes, pachymeningitis cervicalis (a similar form may exist in either the dorsal or lumbar region), neuritis, neuralgia, syringomyelia, lesions of the cauda equina, and circumscribed serous meningitis.

Meningomyelitis can usually be recognized by the early symmetrical distribution of the symptoms and early involvement of the sphincters (p. 387).

Compression myelitis due to caries will be shown by the kyphosis, tenderness, and pain or jarring and the *x*-ray picture.

Tabes, the pains of which may simulate tumor, will be readily recognized by a careful examination (p. 235).

Pachymeningitis may be sometimes impossible to distinguish. The symptoms (p. 212) are usually symmetrical and evidences of segment involvement (p. 367) not so marked.

Neuritis (p. 120) and *neuralgia* (p. 108) will be excluded by careful examination.

Syringomyelia may be impossible to distinguish from intramedullary tumor and in fact is due to a gliomatous formation about the central canal. It must be borne in mind that dissociation of sensation may also occur in extramedullary growths. Hunt states that in such cases, however, tactile sense is diminished when compared with a normal area. Symptoms of lesions of the *cauda equina* are discussed on (p. 204).

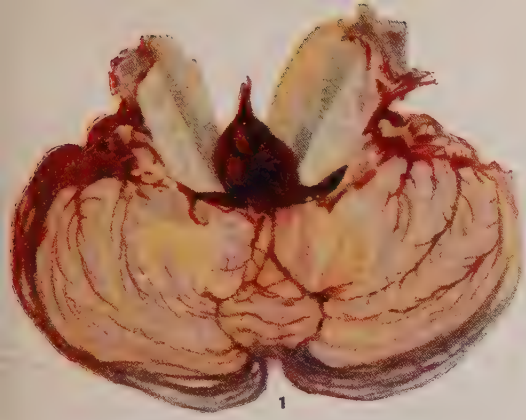
Circumscribed serous spinal meningitis is discussed on (p. 217).

It is important to differentiate intramedullary from extramedullary growths if possible, which it often is not. It has been stated that the appearance of the Brown-Séquard syndrome in an indefinite, atypical form combined with other symptoms of tumors makes it probable that the growth is all or in part intramedullary.

Prognosis.—Unless the tumor can be removed this is fatal, death finally occurring from exhaustion, due to bed-sores and cystitis.

Treatment.—Except in the case of gumma, when active treatment for syphilis is applicable, medical treatment, excepting for the relief of symptoms (see Myelitis), is of no avail. If the tumor is recognized it may in many cases be removed by surgical operation. An exploratory laminectomy should always be done if there is good reason to suspect tumor.[1]

[1] Jour. Nerv. and Mental Dis., April, 1910, p. 244, and May, 1911, p. 169.



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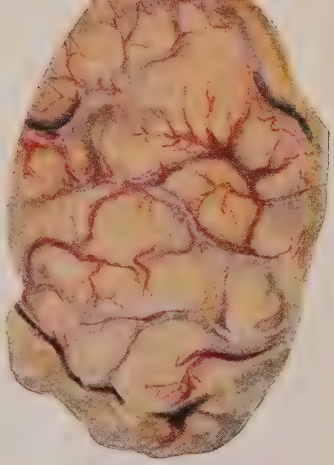

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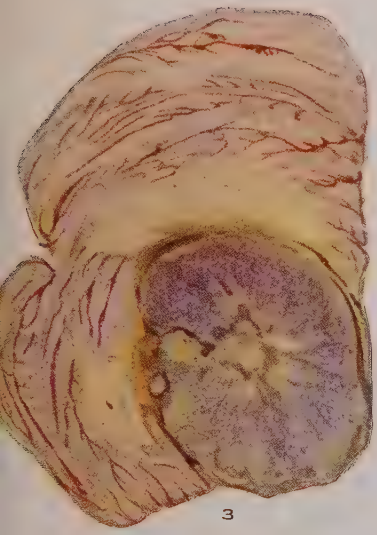

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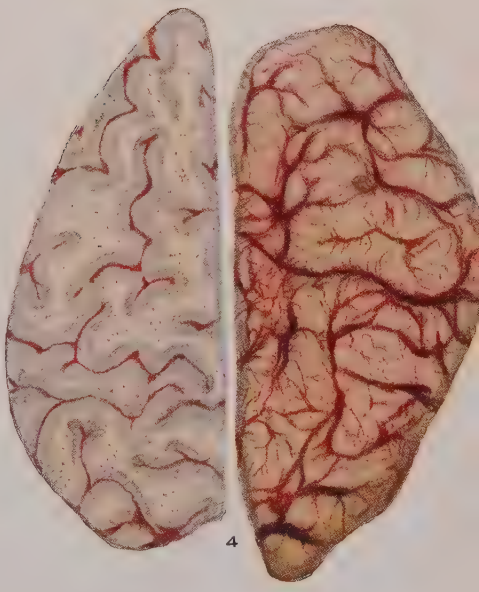

4

1. HAEMORRHAGE IN CEREBELLUM
2. SECTION OF CEREBRAL CORTEX IN ACUTE MENINGITIS
3. GLIOMA OF CEREBELLUM
4. ACUTE CONGESTION OF BRAIN—INTERIOR AND SURFACE

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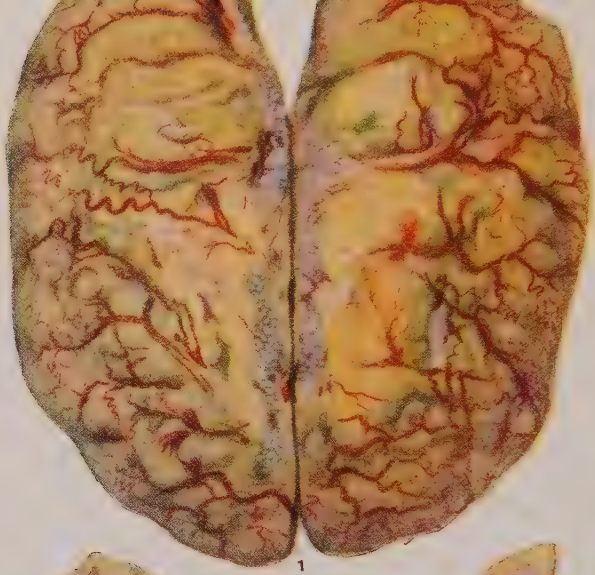

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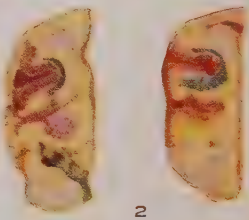

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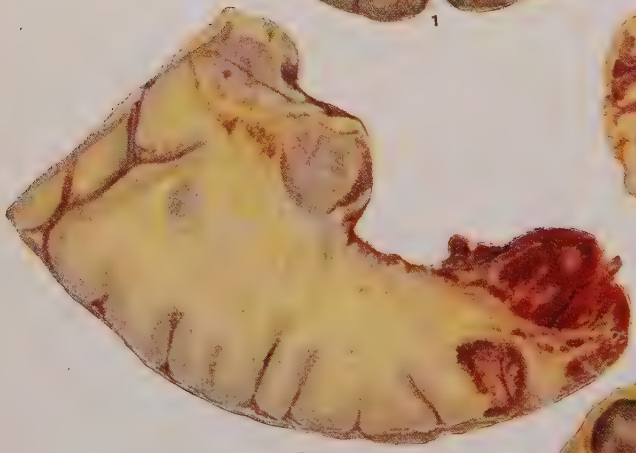

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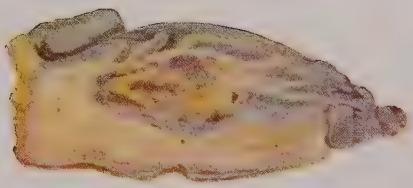

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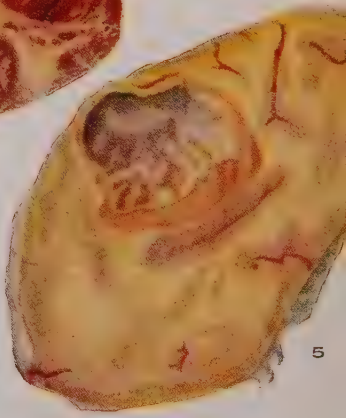

5

1. ACUTE MENINGITIS
2. PUNCTATE HAEMORRHAGES CORTEX OF BRAIN
3. HAEMORRHAGE INTO FRONTAL LOBE OF BRAIN
4. CARCINOMA—BASAL GANGLIA
5. ABSCESS OF BRAIN

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SYRINGOMYELIA

Definition.—This term is applied to the symptoms caused by an acquired *enlargement of the central canal*, or to the formation of new canals in the gray matter of the cord.

Etiology.—The disease is rare. It usually develops early in life, twenty to thirty, especially in men exposed to hard labor. The etiology is obscure. Some cases appear to be caused by trauma, in which instance there is primarily hemorrhage into the gray matter; toxemia, as the infectious diseases; disease of the bloodvessels producing softening; but the majority are probably due to a *congenital dislocation* of some of the *ependymal cells* which are normally found in the central canal, which proliferate and finally degenerate.

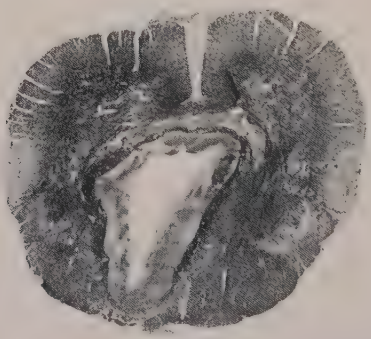
Morbid Anatomy and Pathology.—The usual seat of the cavity is in the cervical region, but it may be limited to other situations or extend throughout the length of the cord, even into the medulla. It may extend irregularly in a transverse direction backward into the posterior horns or forward into the anterior horns, or be merely a concentric dilatation of the central canal (Figs. 112 and 113). The cavity is bounded by neuroglia tissue, which is increased in amount (gliosis), and solid masses of this tissue may be present above and below the cavity. In some cases there is extensive gliosis, with slight or no cavity formation, while in others the cavity is extensive and the amount of gliosis slight. Sometimes the cavity appears to have developed independently of the central canal, but communicates with it. The location of the lesion about the central canal accounts for the dissociation of sensation, which is a prominent symptom. From its location it cuts off the fibers which conduct pain and temperature impressions as they cross over in the anterior commissure, while tactile impressions are conducted direct, principally, at least, in the posterior columns (Fig. 9). Secondary degenerations in the ascending tracts above, and in the descending tracts below the seat of the process, are usually present. Cervical ribs are comparatively frequent in cases of this disease.

Symptoms.—The symptoms come on insidiously and develop slowly, and that which is first noticed is usually a bilateral atrophy and weakness of the muscles of the hands

FIG. 112

Transverse section of the cervical cord in a case of syringomyelia, showing gliosis with extensive cavity formation. The lateral tracts are also seen degenerated. Carmine stain. (From specimen loaned by Dercum.)

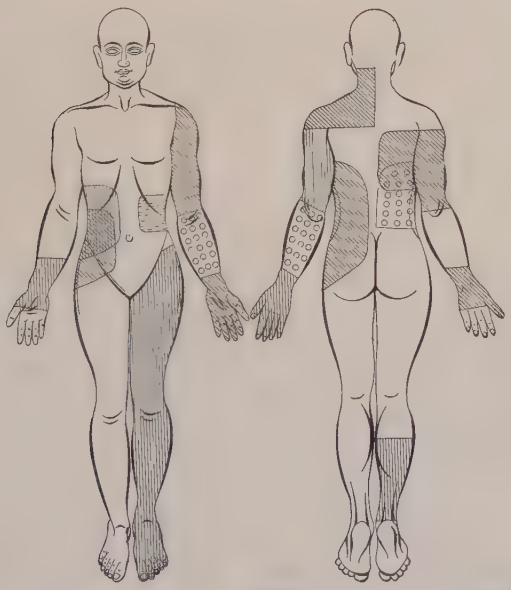
FIG. 113



Transverse section of lumbar cord, showing gliosis with formation of cavity between posterior horns and posterior to central canal, the remains of which can be seen to be distinct from cavity. Degeneration of lateral tracts. Weigert stain.

and arms, resembling in its distribution and clinical features that observed in progressive spinal muscular atrophy (which see). Rarely one side only may be affected. Examination will reveal areas in which there is loss of the perception of pain and temperature with preservation of tactile and muscle sense (dissociation of sensation).

FIG. 114



Sensory chart of patient showing areas of *Thermo-Anaesthesia*, *Analgesia* *Thermo-Anaesthesia and Analgesia*, *Tactile Anaesthesia*, and areas in which the patients answer to tests of temperature showed reversal *Cold-Hot; Hot-Cold.*

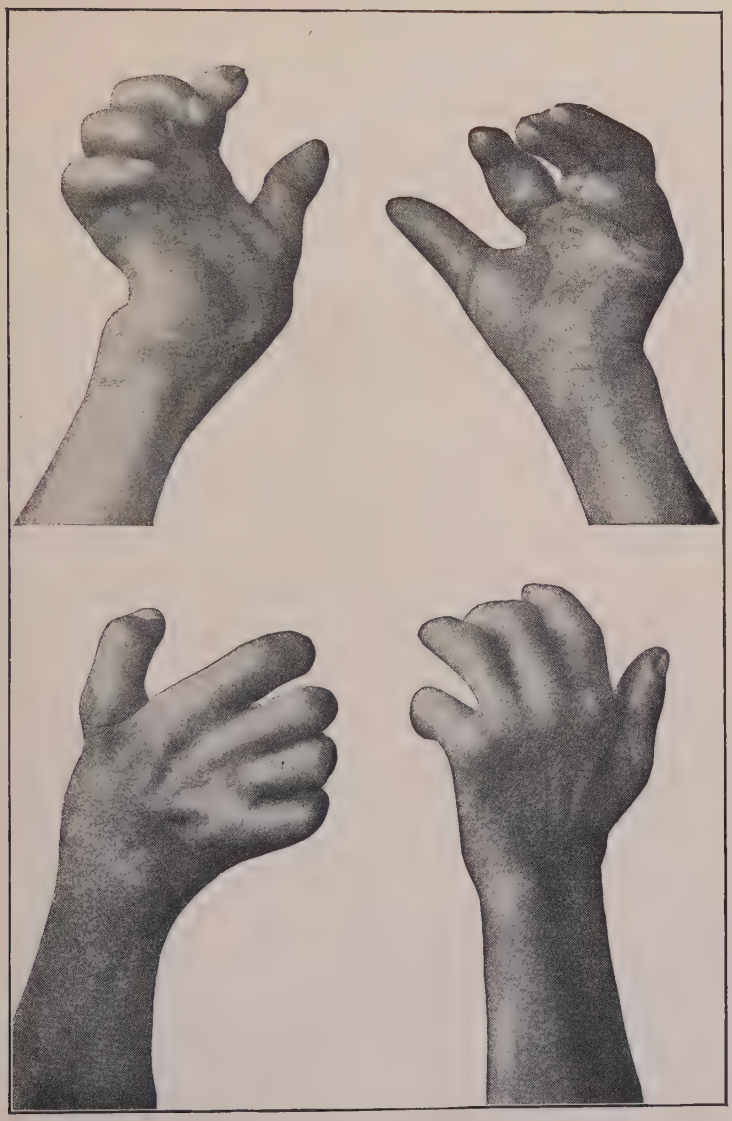
Also temperature sense alone may be lost in certain areas. Areas may be found in which it is not completely lost, but cold may be perceived and heat not, or *vice versa*, or cold may be felt as heat, and *vice versa* (Fig. 114). The loss of sensation occurs in zones, and does not follow the anatomical distribution of the nerves, as in neuritis.

Paresthesias of various sorts and pain referable to the arms, neck, and other parts of the body may be complained of. The sensory symptoms are probably present before the development of motor symptoms, but escape notice by the patient.

When the back muscles become weak scoliosis develops. As secondary degenerations take place symptoms of spastic paraplegia develop, if limited to the pyramidal tracts; of ataxia, if confined to the posterior columns; and of ataxic paraplegia, if both are involved. Trophic symptoms are common, as arthropathies, most common in the arms, fractures of the bone from slight causes, facial hemiatrophy, hypertrophy of the bones of the arms, and skin eruptions and ulcerations. A special type of syringomyelia has been described in which painless felons with bone necrosis occur; this has been known as *Morvan's disease* (Plate VI). Vasomotor disturbances, as lividity, coldness, excessive sweating, etc., also are frequent. If the seat of the lesion is low down (lumbar region), atrophy would occur in the muscles of the legs. If it extends to the medulla, bulbar symptoms occur. The pupils are unequal and contracted, due to injury to the ciliospinal centre. Slight ptosis and nystagmus may also be present. Bed-sores, cystitis, and sphincter paralysis come on late in the course of the disease.

Diagnosis.—At one time dissociation of sensation was thought to be pathognomonic of syringomyelia. We now know that this phenomenon may occur, although rarely, in other disorders which affect the posterior nerve roots, as vertebral caries, cervical pachymeningitis, tabes, spinal tumor, and also in myelitis, multiple sclerosis, disease of the peripheral nerves, and hysteria.

The diagnosis must be made from these and also from progressive muscular atrophy, amyotrophic lateral sclerosis, and anesthetic leprosy. Vertebral caries, tabes, multiple sclerosis, neuritis, and hysteria can be easily distinguished by the other symptoms present in these affections. Cervical pachymeningitis, tumor, and a focal myelitis may be more difficult.



Trophic Changes in the Hands in Syringomyelia in the Form known as Morvan's Disease. (Curschmann.)

The muscles are atrophied. The hands are much deformed. The skin is atrophied. The nails have fallen. The bones are hypertrophied at some parts, atrophied at others. The tip of one finger is eroded.

Cervical pachymeningitis causes more severe pain, and twitchings and spasms of the muscles occur. A *tumor* situated in the gray matter may be indistinguishable. Those springing from the membranes would likely cause more severe pain, and would not produce the development of the peculiar trophic symptoms of syringomyelia.

Focal *myelitis* may also prove difficult, but the more rapid development, with following retrogression of the symptoms, will usually distinguish it.

Chronic poliomyelitis, progressive muscular atrophy, and *amyotrophic lateral sclerosis* are distinguished by the absence of sensory symptoms.

In *leprosy* there is not sensory dissociation, and other symptoms of that disease will appear.

Prognosis.—This as regards cure is bad, no known measures having any influence upon the course of the disease. Intervals of improvement may occur temporarily. The progress is slow, lasting many years.

The **treatment** is purely symptomatic.

CHAPTER XIII

DIFFUSE DISEASES AFFECTING THE BRAIN AND CORD

MULTIPLE SCLEROSIS

Definition.—Multiple sclerosis (also known as insular sclerosis, disseminated sclerosis, and sclérose en plaques) is a chronic affection characterized by localized areas of sclerosis usually scattered throughout the brain and cord, but sometimes limited to either one or the other.

Etiology.—The following have been asserted to be causes: exposure to cold and wet, traumatism, emotional depression, infectious diseases, chronic metallic poisoning, and overwork. A toxemia of some sort is probably the most frequent. It is believed by some authors (Strümpell, B. Sachs) that in many cases, especially those occurring in early life, there is some defect in the original development of the nervous system which acts as a predisposing cause.

It is a disease of early life, the majority of cases occurring previous to the age of thirty, and some even in childhood. It is not very common.

Morbid Anatomy and Pathology.—The sclerotic areas are of irregular size, and may be found in any part of the nervous system, but are more frequent in the dorsal half of the pons, the medulla, in the white matter near the periphery of the cord, and the gray matter near the central canal.

Macroscopically, the sclerotic areas are of a bluish-gray appearance, and may appear either slightly elevated or shrunken and contracted.

Microscopically, there is found thickening of the walls of the bloodvessels, with contraction of the lumen, and the perivascular lymph spaces are filled with cells. Around the

diseased vessels the neuroglia is proliferated, and in the centre of the foci the neuroglia cells are much increased. The myelin sheaths have disappeared; the axis cylinders are apparently normal, excepting in the later stages, when they may degenerate. It is only then that secondary degenerations occur. The ganglion cells also show evidences of degeneration. There is difference of opinion as to the primary seat of the process. In most cases, those due to toxemia, infection, or injury, it is probably the bloodvessels; in others, those due to exhaustion and emotion, the nerve elements are probably first affected. Bruce has called attention to the possibility of the infection being carried in the lymph.[1]

Symptoms.—Owing to the different distribution of the sclerotic areas in different cases the symptoms vary, but there are certain ones sufficiently constant to be considered as characteristic. Usually the first symptom noticed is weakness of one or more limbs, usually both legs, which become spastic. In some cases the gait may be more or less ataxic, resembling that of ataxic paraplegia. There is great increase of the deep reflexes, and absence or diminution of the abdominal reflex occurred in twelve out of fifteen cases studied by Mackintosh.[2] The cremasteric is also often absent; the Babinski phenomenon is usually present. Soon a coarse tremor, developed only when muscular movements are made, appears in the arms (intention tremor), and may afterward involve the head and legs. It is not present when the parts are at rest. It frequently becomes so coarse as to be indistinguishable from ataxic movements. Nystagmus, spontaneous or only developed by movements of the eyes, is a common symptom. Optic atrophy is frequently present, and in a considerable number of cases is confined to the temporal halves of the disk; when present this is a very characteristic sign of the disease. Transient amblyopia with but slight changes in the disk; central scotomata and retrobulbar neuritis may also occur. Paralyses of the motor nerves

[1] Review Neurol. and Psychiatry, July, 1911, p. 361.
[2] Ibid., September, 1906, p. 601.

of the eye, either transient or permanent, are often present. (See Ophthalmoplegia.) Argyll-Robertson pupil occurs rarely. Other cranial nerves, as the fifth, seventh, and twelfth, may also be involved. Speech is usually affected, the patient in most cases articulating in a slow, sing-song manner similar to that employed in scanning a line of poetry (staccato utterance—scanning speech). But other forms of dysarthria resembling those of either paretic dementia, Friedreich's ataxia, or bulbar palsy may occur. Patients may also be subject to attacks of vertigo, epileptiform and apoplectiform seizures. They may be emotional, laughing and crying without cause. Dementia may develop. Muscular wasting sometimes occurs as does also anesthesia, either in small patches or as a hemianesthesia. Various forms of paresthesias may also be met with. Disorders of the sphincters causing incontinence of urine and feces are common.

The *cardinal symptoms* may be tabulated as follows (Peterson):

1. Muscular weakness with rigidity, taking a hemiplegic, paraplegic, or monoplegic form; sometimes with cranial nerve paralysis.
2. "Intention tremor."
3. Exaggerated knee-jerks, wrist-jerks, with ankle clonus, possibly jaw-jerk.
4. Nystagmus.
5. Various symptoms of nervous disturbances, as vertigo, headache, epileptiform and apoplectiform attacks, and mental dulness.
6. Disorders of speech.

While in a typical case the diagnosis may be fairly easy; atypical ones are rather frequent (the "formes frustes" of Charcot). These cases usually resemble some one of the system diseases, as spastic paraplegia or ataxic paraplegia, or a gradually developing hemiplegia (p. 247). Careful study will, however, reveal the presence of one or more symptoms not present in these conditions, such as pallor of the temporal halves of the optic disks, diplopia, sphincter disorders, forced laughter and weeping, vertigo, apoplectiform or epileptiform attacks, paresthesia, etc., which should

excite a strong suspicion of the existence of this disease, especially if the patient be between twenty and forty.[1]

Diagnosis.—It may be confounded with *cerebrospinal syphilis*, which often possesses many symptoms in common with multiple sclerosis. Nystagmus, scanning speech, and intention tremor are rare in syphilis, while in syphilis there are peculiar pupillary symptoms, as absolute immobility, unilateral mobility, and irregular contour of the pupils (B. Sachs).

Syphilis may, however, cause the typical symptoms of multiple scleroses without the development of sclerotic plaques, on the other hand it may cause the development of the plaques without the symptoms.[2] A large increase of the cell content and of the globulin in the cerebrospinal fluid, with a positive Wassermann reaction, would be in favor of syphilis (pp. 98 and 241).

Paralysis agitans is a disease of old age, and the tremor is present when the parts are at rest. Nystagmus and scanning speech are absent.

Hysteria may be confounded. Hysterical symptoms may be present in true multiple sclerosis. In hysteria, however, a true intention tremor is rare, scanning speech, nystagmus, and optic atrophy are absent, and some of the various hysterical stigmata are present.

Cases occurring in early life must be differentiated from the *infantile cerebral palsies*. The paraplegic forms can usually be distinguished by their early onset, occurrence of convulsions, and more marked defect of mental development. The diplegic type presents more difficulty, as nystagmus, peculiar ataxic movements, and speech disturbances may be present. It is rare, however, for more than one of these symptoms to be present in diplegia. The onset is earlier in life, and the paralysis and contractures more marked.

Friedreich's ataxia is distinguished by absence of reflexes, and of optic atrophy, and the presence of more pronounced ataxia of the lower limbs, talipes, scoliosis, and possibly heredity.

[1] Taylor, Jour. Nerv. and Ment. Dis., June, 1906, p. 361.
[2] Spiller and Camp, Univ. Penna. Med. Bull., March, 1909, p. 6.

Hereditary cerebellar ataxia presents more difficulty. The diagnosis may be impossible before autopsy. The Romberg symptom, present in cerebellar ataxia, is rare in multiple sclerosis. The so-called *diffuse* and *pseudosclerosis* also simulate it (see below). Disseminated patches of softening, due to *arteriosclerosis*, may also cause symptoms resembling multiple sclerosis so closely that a positive diagnosis may be impossible. Old age, vascular degeneration elsewhere, and rapid progress would be in favor of softening. *Disseminated myelitis* (p. 381) may also simulate it very closely.

An apoplectiform attack may be the first symptom. Such an occurrence in a young adult, if syphilis and cause for embolism can be excluded, is suggestive.

Prognosis.—The disease is usually incurable, but of slow progression; however, recovery may rarely occur. Remissions also occur which may be of long duration, and even when a cure is not obtained more or less permanent improvement may be hoped for.

The **treatment** is symptomatic. Arsenic and silver nitrate have been recommended. Rest is beneficial. Hexamethylenamin may be tried if an infective origin is suspected.

PSEUDOSCLEROSIS AND DIFFUSE SCLEROSIS

In 1883 Westphal reported two cases in which there was a symptom complex resembling multiple sclerosis, but in which microscopic examination of the brain and cord gave negative results. It was noted, however, in these and subsequent cases that the brain and cord were unusually firm. For a number of years a diffuse sclerosis of the brain and cord has been recognized in those of idiots especially. Lately, however, this condition has been found in brains of those who have suffered from what was apparently multiple sclerosis. Later investigations seem to show that what has been known as pseudosclerosis is really a mild form of diffuse sclerosis.

Symptoms.—The symptoms closely resemble those of multiple sclerosis. Weiss, however, states that nystagmus,

changes in the eye-grounds, sensory disturbances, and localized muscular atrophy do not occur in diffuse sclerosis. He also lays stress upon the occurrence of a general rigidity of the muscles when the patient attempted to move. This was not present in a case observed by the author. The early occurrence of dementia is frequent in diffuse sclerosis, and some cases may closely resemble paresis—in fact, cannot be distinguished.[1]

SYPHILIS OF THE NERVOUS SYSTEM

General Considerations.—The **morbid conditions** of the nervous system due to syphilis may be divided into two groups—*i. e.*, the *specific* and the *parasyphilitic*. The former are specific lesions characteristic of syphilis alone.

The *latter* term is applied to the states in which the lesions are not those characteristic of syphilis, but which are due in some way to the action of the specific poison, which may thus cause disorder of the general health, lessen the vital resistance, affect the development of the fetus and infant, and create organic defects or morbid tendencies. Such affections are (1) those due to acquired syphilis—tabes dorsalis, paretic dementia, neurasthenia, epileptiform convulsions, and mental disorders; (2) those due to hereditary syphilis, viz., juvenile tabes and paresis, hydrocephalus, arrest of development of parts of the nervous system, as possibly some cases of Little's disease, meningo-encephalitis and the hereditary spastic palsies of childhood, arrest of mental development, etc.

The symptoms of the parasyphilitic affections are described elsewhere.

The **specific forms** follow either acquired or hereditary syphilis. In the *former* they may occur at varying periods of time after the appearance of the primary sore. The usual period is between the third and tenth years, but cases have been recorded as occurring during the secondary

[1] Jour. Amer. Med. Assoc., November 11, 1905.

period, even before the chancre was healed, on the one hand, and as late as thirty years after infection, on the other hand.

In the *latter* they may either be present at birth or may appear during childhood. Rarely their appearance may be delayed until late in life. All parts of the nervous system may be affected either singly or collectively.

Morbid Anatomy and Pathology.—The **specific lesions** of syphilis found in the nervous system are:

Gummata, which form definite single or multiple tumors. They are usually attached to the pia, and may either finally invade the subjacent cortex or else only compress it. They appear to spring from the walls of the bloodvessels, being composed of round cells derived from them and of proliferated connective-tissue cells. The walls of the vessels are thickened by these proliferated cells. Gummatous meningitis usually coexists. Occasionally they are attached to the dura, and still more rarely are found in the cerebral substance when they are attached to a vessel. They are not so common in the cord as in the brain, where they are usually found in the cerebral cortex or about the pons.

Gummatous meningitis, in which the membranes are thickened and infiltrated with cells to a greater or less extent. The arteries are also thickened. It is always found in the neighborhood of the large growths above described, but may occur without them. Gummatous meningitis is more apt to involve the dura than is the localized gumma. It is most commonly found at the base.

Diseases of the bloodvessels, which occur as (*a*) *endarteritis obliterans,* in which there is great proliferation of cells in the endothelial and subendothelial tissues, which is greatly thickened, and narrows and sometimes obliterates the lumen of the vessel. The other coats are also infiltrated. This may be also due to other causes.

(*b*) *Gummatous periarteritis,* in which nodular gummata develop in the adventitia of the vessel, producing globular or ovoid swellings. The intima may or may not be involved.

As secondary consequences of bloodvessel disease, we have softening due to thrombosis in or obliteration of the

vessel's lumen, rupture, and consequent hemorrhage, and the development of aneurysm.

Patients in whom the secondary symptoms have been mild are especially likely to develop syphilis of the nervous system, as they usually have not had thorough treatment.

Symptoms.—As all parts of the nervous system may be the seat of the specific lesion, the **symptoms** perforce are varied and widespread. Consequently it is impossible to set up rigid clinical types. But it is a fact that certain parts of the nervous system are more liable to be involved, and, consequently, certain symptom groups are more constantly present. The symptoms are peculiar in that they are often transitory and shifting, appearing suddenly and then disappearing in a few days, others taking their places; that marked remissions may occur, and that paralysis, when it occurs, is apt to be incomplete. The symptoms commonly produced by syphilitic lesions will be briefly detailed:

Cerebral Syphilis.—One of the earliest symptoms is headache, which is constant, severe, and often becomes worse at night. Disturbances of sleep are also common. These may take the form either of insomnia, often accompanied by irritability, or, what is more frequent, excessive somnolence, which may be so pronounced as to cause the patient to fall asleep at his work and consequently unfits him for business. In extreme cases he seems to be in a state of stupor, wandering aimlessly about, seemingly without knowledge of where he is. He seems a restless nocturnal automaton rather than a man (H. C. Wood). From this state he may pass into a profound coma, in which death may occur, but from which recovery is possible.

As has been said, the inflammation is more apt to take place at the base, in which event the cranial nerves in the neighborhood would suffer from pressure or infiltration of their sheaths, causing paralysis. (See Cranial Nerve Palsies.)

When the *optic nerve* is attacked, neuritis is most frequently caused.

The *motor nerves* of the eye are also especially liable to be involved in various degrees. The pupils are frequently

found to be immobile (B. Sachs). Paralysis of the cranial nerves may also be due to a gumma involving their nuclei.

As involvement of the arteries at the base usually coexists, gross paralyses, due to thromboses or hemorrhages, as hemiplegia, aphasia, etc., frequently occur. Thrombosis is more common than hemorrhage. (See Apoplexy.)

When the process involves the convexity, symptoms due to involvement of the cortical centres appear which are focal in character, as Jacksonian epilepsy (p. 439), monoplegia, or hemiplegia, etc. (p. 299). In widespread involvement of the cortex general epileptiform convulsions are often caused. (See Epilepsy.) This may also be a parasyphilitic disease. Diffuse syphilis of the cortex may also cause symptoms resembling those of paretic dementia (which see).

While the symptoms of brain syphilis are usually of gradual onset, they may appear suddenly, with the rapid development of coma, which may or may not be accompanied by convulsions, delirium, and various forms of paralysis sometimes resembling apoplectic attacks.

Neurasthenic symptoms may develop.

Acute forms of insanity may come on at any stage of syphilis, especially in the early stages. *Hypochondria* and *mental depression* sometimes occur, which may be modified or interrupted by attacks of mania or melancholia, and, finally, pass either into dementia or remain constant. These cases are often due to the toxic condition of the blood following the primary infection (p. 526). Maniacal conditions may also occur in the course of mental decay or derangement produced by intracranial syphilitic lesions of the late stage. Frequently such attacks follow convulsive seizures caused by organic syphilitic disease (Mickle).

Syphilis of the Cord.—The symptoms of syphilis of the cord resemble those of syphilis of the brain in their variability and tendency to remissions, there being no one symptom group which can be said to be characteristic. Erb has described a special complex of symptoms which occurs somewhat frequently, and which is known as *Erb's type of syphilitic spinal paralysis.*

This usually begins with slowly increasing weakness and stiffness of the lower extremities, frequently accompanied by paresthesias of the legs, back, and anal region. Actual pain is rare, but may occur as girdle pain on scattered areas in other parts of the body. Anesthesia practically never occurs, but diminution of one or all forms of sensibility may occur. Some incoördination and Romberg's symptom may be present. Disturbance of the bladder function, usually manifested by difficulty in passing water; constipation, and loss of sexual power are usually early symptoms. When fully developed the symptoms are a spastic paralysis of the legs, greater than would seem warranted by the rigidity of the muscles when at rest, for it is then not marked; increased knee-jerks and ankle clonus, difficulty in micturition, constipation, and loss of sexual power, without anesthesia or muscular atrophy.

In these cases the lesion is probably a thrombosis situated in the vessels supplying the lower half of the dorsal cord, and involving chiefly the posterior parts of the lateral columns, less so the posterior columns and horns. The spastic gait, without proportional muscular rigidity and contracture, distinguish this from other forms of spinal paralysis (ataxic paraplegia, lateral sclerosis, transverse myelitis, etc.).

If the lesion consists of a *localized gumma* the symptoms are those of spinal tumor. An area of softening in the gray matter may produce symptoms simulating syringomyelia, or if involving both white and gray matter those of acute transverse myelitis (p. 381). In some instances they simulate those of tabes dorsalis; in others the Brown-Séquard syndrome may be present. Gummata may involve the cauda equina. Spinal symptoms are due to gummatous exudation, and arterial disease, as are those of cerebral syphilis, and may coexist with them.

Syphilis of the Nerves.—The *cranial nerves* may be attacked primarily, especially the optic and motor nerves of the eye, causing optic neuritis and ophthalmoplegia. The *spinal nerves* are more rarely involved, unless it is involvement of their roots by a syphilitic exudation, when the symptoms of nerve-root irritation and compression will be caused (p. 216).

Diagnosis.—If obtaining a history of previous syphilitic infection is not possible, the evidences of it upon the person, or a history on the part of the wife of frequent miscarriages, stillborn children, or deaths in early infancy, are suggestive. It is important to remember the frequent mildness of the secondary symptoms in those who develop nerve syphilis. The diagnosis is based on the multiplicity of the lesions, their peculiar progress—*i. e.*, their fugacious character and frequent remissions—and the general subacute character of the affection. In the absence of hysteria, any indefinite and apparently disconnected series of nerve accidents is of very urgent import (H. C. Wood). The age of the patient is suggestive. Apoplexy, not due to embolism, or following one of the infectious fevers, occurring in early adult life, is most frequently due to syphilis; and epileptiform attacks occurring after thirty-five are frequently so caused. (See Epilepsy.) Cases of sudden coma due to syphilis present great difficulty. All other causes of coma must be eliminated. A pronounced rise of temperature or conjugate deviation tells against, while ocular palsies or partial paralysis of any character argues in favor of a specific origin (H. C. Wood).

Previous to the development of the more permanent symptoms a previous history of headache, insomnia, or somnolence, transient double vision, or other evidence of a transient palsy, may often be obtained. While cord syphilis may simulate any of the organic diseases of the cord, some atypical feature will generally be present, as absence of rigidity and contractures in spastic paralysis, absence of Argyll-Robertson pupil in cases simulating tabes, etc. Isolated paralysis of any of the cranial nerves points to syphilis in the absence of other apparent cause. B. Sachs lays stress upon the frequent occurrence of immobility of the pupil. A *marked increase* of globulin and lymphocytes (p. 418) with a positive Wassermann reaction with the blood points to syphilis. In tabes the reaction is usually negative with the fluid but may be present with the serum. In paresis it is usually present with both, but may be positive with the fluid and not present with the serum. Often, however,

our diagnosis depends for certainty upon the fact that symptoms succumb to antisyphilitic medication (see p. 98).

Prognosis.—While, on the whole, the prognosis in cases of syphilis in the nervous system is favorable, it is not always so. If the nerve elements are destroyed by the lesion, as, for instance, by a cerebral hemorrhage, the prognosis is no better than that of similar conditions due to other causes. Also, while a gumma may be made to disappear by treatment it may leave a scar in the nerve tissue that will cause permanent symptoms. Hence there should always be some reservation. In any case the prognosis becomes unfavorable in proportion to the length of time that the symptoms have persisted and the age of the patient. A disappearance of the lymphocytes from the cerebrospinal fluid under treatment is a good omen. Death may occur during a syphilitic convulsion or attack of congestion, or from the involvement of some vital centre.

Treatment.—In cases of coma, salvarsan, if there is no contraindication, as myocarditis and disease of the liver and kidneys, is the most efficacious remedy if it can be obtained quickly. If not, mercurial injections or inunctions should be used. If the diagnosis is not immediately made measures similar to those employed in apoplexy may be used. Salvarsan is the remedy to use in all urgent, acute cases. Its employment should be followed by a long-continued course of mercury and iodide of potassium in large doses. If it cannot be administered, mercury should be used either by injection or inunction until active symptoms are relieved; it may then be given internally with iodine in some form. For the hypodermic injection corrosive sublimate in doses of one-twelfth of a grain in distilled water is a convenient form, and should be injected deep into a large muscle, as the gluteals. Combined with these measures the employment of tonics, nutrients, and symptomatic treatment is advisable. If cortical focal symptoms are present and do not soon yield to internal medication an exploratory operation is justified, and if a gumma be found it should be removed (p. 362).

PARALYTIC DEMENTIA

Synonyms and Definiton.—This disease is known also as progressive general paralysis, paretic dementia, general paresis, paresis, general paralysis of the insane, chronic diffuse meningo-encephalitis, chronic peri-encephalitis. By the laity it is often termed softening of the brain. It is a disorder characterized mentally by a progressive enfeeblement associated with either exalted or depressive delusion, or both alternately. Physically there is also progressive enfeeblement, often associated with ataxia.

Etiology.—Paralytic dementia may occur at any time from childhood to old age; but it appears chiefly between the ages of thirty and fifty-five. It more frequently attacks the male sex. According to Mickle, they are attacked about four times as often as the female. A neuropathic heredity may act as a predisposing cause. Probably the only cause of true paresis is syphilis, Juvenile cases may be due to hereditary syphilis. It is a parasyphilitic disease (p. 407). Causes which may help develop the disease in one who has had syphilis are alcoholism, excessive venery, prolonged mental overstrain and excitement, prolonged and exhausting physical work and head injuries. Injuries to the head, while put down by some as a cause, is a doubtful etiological factor unless the patient has had syphilis, in which event it may probably hasten the development of the disease.

Morbid Anatomy.—The *macroscopic* changes are: Increase of the cerebrospinal fluid, edema of the pia, chronic leptomeningitis, the membranes being thickened and opaque and adherent to the cortex. In about 50 per cent. of the cases pachymeningitis hæmorrhagica interna is also present (p. 211). The ventricles are sometimes found to be dilated and distended with serum, while a granular condition of the ependyma is a constant and characteristic change. There is diminution in the weight of the brain, and the convolutions are atrophied and the fissures patulous, especially in the frontal and parietal regions. The cortex in color is either extremely pale or is a marked rosy tint, due to congestion of

the small bloodvessels. Areas of softening or hemorrhage, due to arteriosclerosis, are frequently found scattered throughout the brain. In consistency the cortex may be either softer than normal, or firmer, due to sclerosis. These different conditions may be found in the same brain, the latter probably being the ultimate result of the former. Sclerotic changes are also found in the pons, medulla, and frequently in the posterior and lateral columns of the cord. As has already been said, tabes may either follow or precede the development of paretic dementia, the two diseases being certainly closely related, if not the same disease.

Microscopically, changes are found in the walls of the capillaries, which are thickened and present a granular appearance, due to increase in the number of nuclei. The adventitia of large vessels is infiltrated more or less with small round cells. The perivascular spaces are dilated and contain these cells. Leukocytes and red blood cells may also be found.

The ganglion cells show evidences of degeneration, evidenced by chromatolysis, loss of processes, and disintegration of the nucleus.

The white fibers are more or less degenerated, especially the tangential fibers. There may also be an increase in the number of neurogliar or spider cells.

Symptoms. — In rare instances paralytic dementia may begin suddenly with an epileptiform or apoplectiform seizure, which is followed by a rapid development of the characteristic symptoms. In the great majority of instances the mode of onset is gradual, constituting a *prodromal stage* of greater or less duration. There will be noticed changes in the character of the patient; for instance, from a man of good habits and regular mode of life he becomes intemperate, licentious, neglects his business, and spends money faster than his income warrants. He is moody and irritable and spells of depression may also occur. Failure of memory, insomnia, and a ravenous appetite are frequent in this stage. While these symptoms of themselves are not sufficient to make the diagnosis of paralytic dementia, they should awaken strong suspicions when they are present in a person

liable to develop the disease. Other cases may have presented symptoms of a neurasthenic nature for some time before characteristic symptoms develop.

The symptoms of the *second stage*, or developed disease, are both mental and physical. The usual mental condition is one of a more or less marked feeling of exaltation; he becomes restless and irritable, insomnia is present, and the so-called delusions of grandeur develop. For instance, he believes himself to be possessed of untold wealth, to be the strongest man in the world, a great pugilist, or is going to engage in some enormous business enterprise.

In place of delusions similar to those mentioned there may be merely a feeling of well-being and indifference. When asked how he is, the answer will always be "first rate," or that he "feels well." While he may be a poor man, the fact that he is unable to work and support his family does not worry him.

In some cases, instead of the exalted condition, the patient becomes depressed, hypochondriacal, and may apparently appreciate and worry about his condition. In other cases there may be alternate periods of exaltation and depression. Hallucinations are rare and usually occur in the depressed cases and are of a hypochondriacal nature. Rarely the mental symptoms may be either of a maniacal type, delusions of persecution may predominate or Korsakow's psychosis (p. 534) may be simulated. Periodic outbreaks of great excitement may occur, and exaltation, depression, and a comparatively normal state may alternate as in circular insanity.

The *physical symptoms* are of great importance in arriving at a diagnosis. One of the earliest to be noticed is disturbance of the speech. This is slow, interrupted, and slurred, resembling somewhat that of a drunken man. Special difficulty is encountered in pronouncing labials and linguals and the inability to pronounce test-phrases and sentences, as "truly rural," "legal intelligencer," "Peter Piper picked a peck of pickled peppers," etc., is among the early evidences of the disease. Tremor of the muscles of the face, especially of the lips and of the hands, will be noticed. Owing to the

latter condition, writing becomes difficult or impossible. The tongue is also tremulous and is protruded with difficulty, and when protruded cannot be kept so. The expression of the face is stolid and the facial lines are smoothed out. General muscular weakness and inability for exertion will be found, although the patient will assert the contrary. The knee-jerk may be either increased, normal, diminished, or lost. The pupils are usually unequal, and present the Argyll-Robertson phenomenon. Primary optic atrophy also may occur. There is often more or less marked ataxia. In fact, all the characteristic symptoms of tabes may either follow or precede the development of paresis. Examination of the cerebrospinal fluid obtained by lumbar puncture shows an increased number of lymphocytes (p. 418), plasma cells, and globulin. In a large majority of cases[1] the Wassermann reaction is present with both serum and cerebrospinal fluid. Marie states that in early cases it may be present with the blood but not with the serum.

During the progress of the disease, epileptiform, apoplectiform, or hysteroidal seizures may occur. The former are usually mild, and the convulsive movements are limited to the face and arm of one side. They may, however, be severe, and sometimes cause death. The apoplectiform seizures may consist of coma, facial congestion, elevated temperature, and temporary hemiplegia; or transient paralysis may occur without loss of consciousness.

Remissions in the symptoms may occur, and it may be thought that recovery is going to ensue; but they return and progress until, if death does not occur, the *final stage* is reached.

In this the dementia becomes absolute, the patient becomes weaker and weaker, and finally bedridden; bed-sores and paralysis of the bladder and rectum develop, and finally death occurs.

Diagnosis.—Paretic dementia may be confounded with neurasthenia; the mental changes produced by chronic alcoholism, plumbism, and diabetes; cerebral syphilis; chronic

[1] In Plaut's series of 153 cases, in 131 it was present with both, in 16 with the spinal fluid alone, and in 6 with the serum alone. See also p. 241.

mania with delusions of grandeur, multiple sclerosis; bulbar palsy; and dementia associated with organic diseases of the brain—viz., tumor and apoplexy.

It *resembles neurasthenia* only in the early or prodromal stage, when the symptoms may be very similar. The occurrence of such symptoms in a previously healthy man who has been exposed to any of the etiological factors of paresis should awaken suspicion, especially if he becomes absent-minded, negligent of his business, irritable, exhibits loss of attention, concentration, and judgment, and increase of sexual power.

Chronic alcoholism may present many of the symptoms of paresis—*i. e.*, tremor, thick speech, and dementia. Often it is only by the improvement that occurs after the withdrawal of the alcohol that the differential diagnosis can be made (p. 533). Cerebral disorder due to *chronic lead poisoning* may imitate closely the facial expression, tremor, etc., of the paretic. A history of exposure to the poison and the presence of other evidence of lead poisoning (blue line, neuritis) would distinguish the former. Examination of the cerebrospinal fluid and performance of the *Wassermann test* will be of value in eliminating the above conditions.

Cerebral syphilis may simulate paresis more closely than any other affection. The distinction may often be impossible except by noting the effects of specific treatment. Mott states that in pseudoparesis the lymphocytes in the cerebrospinal fluid disappear under antisyphilitic treatment but in true paresis they do not. The presence of plasma cells in the fluid and the Wassermann reaction being obtained with both it and the blood are indicative of paresis. The cells are in much greater numbers in the fluid in true syphilis (95 to 451 per c.mm.) than in paresis (8 to 78 per c.mm.). The following differential points are abstracted from a paper on this subject by Patrick:[1] When the symptoms appear at a remote period (after ten years) after infection the disorder is probably paresis. If they appear

[1] Points of Distinction between Cerebral Syphilis and General Paralysis of the Insane, New York Med. Jour., vol. lviii, p. 256 et seq.

within a few years (one to ten years) after infection the probabilities are in favor of cerebral syphilis. In paresis the early or prodromal symptoms are disturbances of the higher mental functions. The higher faculties being first attacked, the mental acts of routine existence that have become nearly automatic are impaired last; thus, a man may be able to conduct his own business for some time after he has exhibited much impairment of the ability to perform other mental acts. In cerebral syphilis these symptoms are usually somatic, as paralysis in various situations, usually focal, sensory disturbances, etc. In cerebral syphilis, therefore, there are apt to be symptoms added to the clinical picture that are not usual in paresis.

Severe headache is practically always present in syphilis; practically absent in paresis. Some headaches may occur in the early stages of paresis, but not the characteristic pain of cerebral syphilis.

Pains in other parts of the body, worse at nights, are characteristic of syphilis, and against paresis.

Marked anesthesia or paresthesia is much more frequent in syphilis.

Paralyses of any of the cranial nerves, below the third, are in favor of syphilis. While the third nerve may be also affected in paresis, there are differences which assist in the diagnosis—viz., Argyll-Robertson pupil is strongly indicative of paresis. Either complete immobility of the pupil (internal ophthalmoplegia) or external ophthalmoplegia is indicative of syphilis. Association of paralysis of the third nerve with that of other cranial nerves is indicative of syphilis.

The physical weakness, tremor, etc., occurring in paresis are generalized. Any symptoms pointing to a localized or focal brain lesion would indicate syphilis. When localized paralyses do occur in paresis they usually follow one of apoplectic attacks, which occur in the course of the disease, and in the great majority of instances are transient.

Primary optic atrophy is a symptom of paresis. Optic neuritis and consecutive atrophy are indicative of syphilis.

Persistent yawning, if there are no signs of bulbar disease, indicates paresis.

Marked loss of weight often occurs in the early stages of syphilis. In paresis, excepting in the advanced stages, this is very rare.

The presence of polydipsia, polyuria, or vomiting of the cerebral type would most likely be due to syphilis. The mental state following an epileptiform seizure may be of assistance. In paresis this is always distinctly worse; but in syphilis such is not usually the case. If in the early stages there are mental eagerness and restlessness, an hyperactivity without hyperacuity, increase of sexual desire and power; or if, instead of the above, there is a long period (one to five years) of prodromal symptoms resembling neurasthenia, the disease is paresis. Either marked delusions of grandeur or exaggerated hypochondriacal delusions very rarely occur in cerebral syphilis. The condition of contentment, and "being pleased with one's self," spoken of under Symptomatology, is more common in syphilis, but is also frequent in paresis.

Chronic mania would be distinguished by a history of previous acute mania and the absence of the physical symptoms characteristic of paresis. Acute excitement (delirium) may occur as an episode in the course of paresis, but attention to the history and physical symptoms will reveal the true nature of the illness; the same may be said of attacks of depression which sometimes occur. The egotism and exaltation often found in *paranoia* can hardly be confounded if attention is paid to the physical symptoms. *Multiple sclerosis, bulbar palsy,* and *dementia* due to any other cause, could hardly be confounded with paresis if careful attention is paid to the history of the mode of onset and the symptoms carefully investigated.

Prognosis.—Paretic dementia usually runs its course in from three to five years, the ultimate result being death. Rarely, cases may last longer than this.

Treatment.—The effects of antisyphilitic treatment may at first be given a fair trial if the Wassermann reaction is present with the blood only and the changes in the cerebrospinal fluid found in syphilis are present (pp. 412 and 418), otherwise nothing but symptomatic treatment is of any use, and committal to a hospital should be advised.

AFFECTIONS OF THE NERVOUS SYSTEM DUE TO ARTERIOSCLEROSIS

The importance of vascular degeneration in the causation of lesions of the nervous system is being more and more recognized. Some have been mentioned in other places (pp. 40, 112, 120, 130, 322, 381, 389, 406, 436, 445, 494, and 499). A symptom group is often presented in which the patient may complain of slight headache, vertigo, poor station, laughing and crying without cause, expressionless face, and a peculiar gait, consisting of very short, shuffling step, with the feet wide apart. The knee-jerks are usually increased and the Babinski phenomenon may or may not be present. Mental failure is frequently associated.[1] Symptoms resembling those due to disease of the nervous system have been caused by the obliteration of a vessel in one of the limbs, as in the case reported by Burr and Camp,[2] in which an old hemiplegic developed an obliterating arteritis in the vessels of his good leg, which caused a paralysis of that leg and a consequent triplegia. Such cases may be preceded by intermittent lameness (p. 499), and, if suddenly developed, gangrene occurs.

Degenerations of various tracts causing either focal or diffuse symptoms may be due to small areas of softening.

It is important to remember that arteriosclerosis of the vessels of the nervous system may be present when it is not apparent elsewhere.

[1] Collins, Jour. Nerv. and Ment. Dis., December, 1906, p. 750.
[2] Amer. Jour. Med. Sci., June, 1905.

CHAPTER XIV

GENERAL AND FUNCTIONAL DISEASES

PARALYSIS AGITANS

Etiology.—This disease, also known as Parkinson's disease and *shaking palsy*, usually makes its appearance between the ages of forty and sixty-five years. Cases have been reported in which it appeared much earlier, but these are rare. It is more common in males. A neuropathic heredity can be traced in most cases. Fear, anxiety, exposure, overwork, grief, and trauma have been given as exciting causes.

Morbid Anatomy.—While paralysis agitans has been classified under functional diseases, investigations have tended to show the occurrence of a fairly constant number of anatomical changes, principally in the spinal cords of these patients. They consist of congestion and dilatation of vessels in the gray matter, atrophy and pigmentation of the cells and an increase of interstitial tissue. The central motor neurone appears to be most at fault (Dana). It has been stated by some that the disease is dependent upon an affection of the muscles, due to a general toxemia, possibly dependent on the disturbance of the functions of the parathyroid gland.[1] In four cases Roussy and Clunet[2] found them enlarged and presenting the histological appearances of hyperfunctioning.

Symptoms.—Usually the first symptom noticed is muscular rigidity, affecting principally the flexors of the arms, head, trunk, and legs, which produces the characteristic position (Fig. 115)—*i. e.*, the head and body are bent forward, the fingers flexed as a whole on the metacarpal bones, the forearms flexed on the arms, and the knees slightly bent. The symptom may commence on one side,

[1] Camp, Jour. Amer. Med. Assoc., April 13, 1907.
[2] Soc. de Neur. de Paris, February 24, 1910.

which may be affected for some time before symptoms appear upon the other. Rarely the extensors of the neckare affected, causing the head to be drawn backward. Cramps may occur and there is a sense of stiffness. Muscular movements are slow. The deep reflexes are not increased. The facies is characteristic, the lines of expression becoming smoothed out, the eyelids widely opened and rarely winking, the appearance resembling that of a face covered by a mask. The gait is peculiar, the steps being short and shuffling, often with a hesitancy in starting; often, also, there is a tendency to run suddenly forward, the patient appearing to hasten his steps in order to keep up with his body, which is held advanced beyond the perpendicular (festination). The tremor either appears after the rigidity or about the same time. It usually commences first in one hand and arm, then after a greater or less period of time invades the leg, and finally involves the other side. The tremor is slow, and the patient seems to be constantly rolling some small object between the thumb and fingers. It is worse while the limbs are at rest, and usually ceases for a few moments after voluntary movements are begun. It ceases during sleep. The muscles of the neck and head are but rarely

Fig. 115

Photograph of a case of paralysis agitans, showing the attitude, the position of the hands, and the facies.

affected. Motor weakness is not a marked symptom, and usually is not evident at all until the disease is of long standing.

The sensory symptoms consist of dull pains in the limbs and a feeling of heat. Vasomotor disturbances, evidenced by increased surface temperature, sweating, and flushing, may be present. The mental faculties, if aroused by some stimulation, are not much, if at all, lessened; but ordinarily the patient thinks slowly, is apathetic, and appears to take no interest in his surroundings. The speech becomes hesitating and monotonous.

The appearance and attitude of a patient with paralysis agitans are well shown in Fig. 115. The course of the disease is progressive but slow, lasting sometimes as long as forty years. The patient may eventually become helpless and demented. Cases occur in which the tremor is absent, the diagnosis then depending upon the peculiar rigidity, attitude, facies, etc.

Diagnosis.—The diagnosis is usually not difficult. The tremor possesses a feature peculiar to that of this disease in the temporary cessation which occurs after voluntary motion. This alone will serve to distinguish it from *multiple sclerosis*, which in addition differs in the presence of increased knee-jerks, ankle clonus, nystagmus, peculiar speech, etc. *Senile tremor* occurs in the very old, and usually affects the head first. *Posthemiplegic tremor* is distinguished by a previous history of an apoplectic attack and the presence of hemiplegia. Other causes of tremor and their characteristics are given on p. 40.

Prognosis.—The disease in incurable, but may last a long time, and remissions sometimes occur.

Treatment.—The patient should lead a quiet life with plenty of fresh air and plenty of simple, nutritious food. Tonics, such as quinine, arsenic, and mineral acids, are helpful. Lukewarm baths and electricity in the form of the constant current, applied daily, have also been recommended. The most useful drug for relieving the tremor is hyoscine hydrobromate, in doses of gr. $\frac{1}{100}$ t. i. d., and increased. Codeine is also useful for this purpose. Gowers

recommends cannabis indica and arsenic. Parathyroid gland in doses of gr. $\frac{1}{2}\frac{1}{0}$ of the powdered gland two to four times daily has been recommended.[1]

CHOREIFORM AFFECTIONS

These are also termed the *myoclonias*, from *myoclonia*, which means a disease characterized by muscular twitching. *Myoclonus* means a twitching muscle.

Sydenham's Chorea. — **Definition.** — Sydenham's chorea, infectious chorea, infectious myoclonia, chorea minor, St. Vitus' dance, St. Anthony's dance, is an acute functional disease, occurring principally in children, and is characterized by irregular and incoördinate muscular contractions and twitchings, which are ordinarily beyond the control of the will and cease during sleep. There is present in most cases more or less psychical impairment (Sinkler).

Etiology.—A neuropathic history in the parents or relatives can usually be traced. The majority of cases occur between the ages of five and fifteen. It may occur younger. Sinkler mentions a case which has persisted since birth, due to a fright which the mother received during pregnancy. It may also come on in adult life or old age, when it is known as senile chorea. It is more common in girls than boys. Chorea is more common in the city than in the country, and occurs most frequently in the spring and summer.

It is probably an infectious disease of bacterial causation, although the exact nature of the infection is unknown. There is evidently some relationship between chorea and acute articular rheumatism, as it frequently follows that disease. It may also follow other infectious diseases, especially scarlatina and tonsillitis. Pregnancy is a common cause in adults. Fright seems to be a rather frequent exciting cause. Reflex irritations, as eye-strain, adenoids, etc., while they may aggravate the symptoms when they exist, are not causes. Overwork and study may have some influence in exciting an attack.

[1] Berkley, New York Med. Jour., November 23, 1907, p. 974.

Morbid Anatomy and Pathology.—The lesions found in Sydenham's chorea are cerebral hyperemia, with dilatation of the vessels, periarterial exudations, some thickening of the intima of the small arteries, erosions, spots of softening, minute hemorrhages, and emboli. They are found in the motor tract, particularly the lenticular nuclei and thalami, the gray matter of the cortex and spinal cord. In chronic cases perivascular dilatations and increase of connective tissue is found. In the heart evidences of endocarditis may be present.

Symptoms.—Usually the first symptom noticed is a general restlessness and inability to sit still. Close observation will also show twitching of the fingers of one hand, a shrugging of the shoulder, or drawing of the mouth at intervals. If the child attends school, it will be noticed that the writing deteriorates, and that the pencil or other objects grasped are dropped from the hand. The child becomes peevish, irritable, and emotional. Dull pains in the joints are often complained of and headache may be present. As the disease advances, the movements (the characteristics of the choreic movement are described on page 41) become more marked and generalized, but do not always involve all the muscles. In mild cases they can for a time be controlled by the will, and are not severe enough to prevent the patient from performing voluntary movements, such as dressing, undressing, and feeding himself. In the severe cases the movements become general, every muscle is in violent action, so that it may be impossible for the child to sit still. In some instances it is necessary, in order to keep the patient from falling out of bed, to surround it with high-padded sides; or to tie him by means of a sheet passed around the body and fastened to the bed. Owing to the irregular contractions of the muscles of articulation, speech is interfered with. In the milder cases these may consist of talking in a thick and indistinct manner and a spasmodic jerking out of words, while in very severe cases the patient cannot articulate at all.

Contractions of the laryngeal muscles sometimes cause involuntary explosive sounds to be uttered. Rarely, the

choreic movements may be confined to these muscles, a condition known as *laryngeal chorea*. The face, in the intervals between the facial movements, assumes a blank and vacant expression. The muscles of respiration, especially the diaphragm, are also sometimes affected, causing irregular respirations—sometimes shallow, sometimes deep, with occasionally a few rapid, shallow respirations and then a long sighing one. The heart's action may also be irregular. During sleep the movements usually cease, but the sleep is restless and enuresis is sometimes suffered from. The patient, no matter how severe the movements, seldom complains of being tired.

Muscular weakness may exist, shown by dragging the leg in walking or a weakening of the grasp. This is seldom pronounced, but in rare instances the paresis is a more marked symptom than the movements (*paralytic chorea*).

Heart murmurs are frequent. These, in the majority of cases, are hemic, depending upon the anemic condition often found in these cases. The murmur is usually soft, systolic, and heard at the apex. Endocarditis is a frequent complication of chorea; it is usually, however, of a mild type. Of 110 cases examined by Osler, two years and longer after an attack of chorea, 64 presented some sign of organic heart disease. Pericarditis is an occasional complication. During the attack the temper is irritable, there are frequent hysterical outbreaks, and inability to study or apply one's self. In rare instances depression or excitement with hallucinations may occur. Cases of violent choreic movements, usually occurring in adult women, associated with high temperature, great mental excitement, and delirium, are known as *chorea insaniens*. Convulsive attacks very rarely occur. The knee-jerks are sometimes absent, but are capable of reinforcement. Trophic changes are shown by a tendency of slight scratches to become inflamed and wounds heal slowly.

Various skin eruptions, as herpes, various forms of purpura, erythema nodosum, and the formation of subcutaneous fibrous nodules, such as are associated with rheumatism, are sometimes observed.

The attack usually reaches its maximum in about two

weeks, and its duration varies from three weeks to three months or longer, the average being ten weeks. Sometimes cases become chronic and continue during life. Remissions during an attack may occur, and patients who have had one attack are liable to have future ones.

Diagnosis.—The diagnosis is usually not difficult.

Hysterical chorea, or Bergeron's electric chorea, may be mistaken. This is almost always seen in young adults. The movements are more rhythmical, and frequently they consist of sudden jerks, like those caused by electric shocks. They increase in severity when the patient is under observation. Other stigmata of hysteria will be present.

Athetoid and *choreiform movements* occurring in cases of infantile cerebral palsies may simulate chorea, especially when the paralysis is not marked. The history of the onset, rigidity of muscles, increased reflexes, rhythmic character of the movements will make the diagnosis clear. In tics (p. 430), movements are confined to a certain set of muscles, usually those which take part in some particular movement, and they are more rhythmical than those of chorea. The characteristics of hereditary chorea, tic convulsif, and myoclonus are described on pages 429, 432, and 433.

Senile or *adult chorea*, if it becomes chronic, may be confounded with hereditary chorea. The features in which the two differ are given under the description of that disease.

Prognosis.—As regards life, the prognosis is good. Cases of chorea insaniens may die. The average duration is from six to ten weeks. The disease sometimes becomes chronic. If it has lasted over six months it may be said to be so. It is more apt to do so in cases occurring late in life.

Treatment.—All sources of reflex irritation, as refraction errors, nasal disease, adherent prepuce, must be sought for and removed if found. The child should be removed from school and all sources of excitement avoided. The diet must be simple and nutritious; tea, coffee, pastry, candy, etc., being avoided. In severe cases absolute rest in bed is essential, and with this gentle massage once or twice daily is beneficial. The patient should be bathed daily. Warm

wet packs often have a soothing influence. In milder cases, instead of absolute confinement to bed, the child may be allowed to be up and in the air part of the day.

Arsenic, in the form of Fowler's solution, is the most efficacious drug. It should be given in ascending doses until the toxic effects of the drug are manifested, when it may be stopped for a day or two, and then continuing with the maximum dose that will not produce these symptoms. Large doses of quinine, as recommended by H. C. Wood, are sometimes useful. Antipyrine, cimicifuga, and monobromide of camphor may prove useful in some cases. The hypodermic injection of small doses of apomorphine, gr. $\frac{1}{40}$, has also given good results. Along with these drugs, tonic and nutrients, as iron, small doses of strychnine, cod-liver oil, etc., are usually indicated. In the severe cases bromides, chloral, or hydrobromate of hyoscine may be required to quiet the movements and promote sleep.

Hereditary Chorea.—Synonyms and Definition.—This disease, also known as chronic progressive chorea, Huntingdon's chorea, and degenerative myoclonia, occurs in successive generations of a family, making its appearance in the victims in most cases at about the ages of thirty to forty, and is characterized by peculiar general rhythmic movements and mental deterioration.

Morbid Anatomy and Pathology.—The principal macroscopic change is an atrophy and thinness of the cortex, and in some cases evidences of meningitis. Microscopically, evidences of a slowly progressive degeneration of the ganglion cells is found most marked in the Rolandic region. Slight increase of the neuroglia, distention of the perivascular spaces, and slight disease of the bloodvessels occur, and slight degenerations in the pyramidal tracts. The cause is probably a premature death of the cell, owing to an inherent weakness (abiotrophy).

Symptoms.—Previous to the appearance of the movements, the patient may suffer from nervousness, weakness, and loss of ambition; and difficulty is noticed in performing fine movements, as writing. The movements usually first appear in the upper extremity, resembling those of ordinary

chorea. In time they become general, involving both arms and legs; and speech, which is slow and difficult, and swallowing are interfered with. When fully developed, the movements differ from those of chorea, being more rhythmical and more extreme, whole groups of muscles being brought into action. The gait is characteristic, the patient taking a few steps naturally, then takes a long step with one leg, bringing up the other quickly to it and making one or two hops, which gives the appearance of dancing. Sometimes there is a tendency to stand in one position for a length of time. In the early stages the movements may be controlled by the will. In the later stages effort to do so increases them. Mental disorder is found in all cases. Usually it appears late, but sometimes it precedes the motor symptoms. It usually consists of mental depression, with suicidal tendencies, delusions of persecution, with the development of dementia.

Diagnosis.—*Friedreich's ataxia* appears in early life; the movements are not choreic, but ataxic; the knee-jerks are absent; nystagmus is present. In *multiple sclerosis* the movements cease while the patient is at rest.

Prognosis.—The disease is incurable, but it may last a long time before death occurs.

Treatment.—The treatment consists of increasing the general nutrition as much as possible and relieving symptoms as they arise. Arsenic and nitrate of silver may be tried. Collins advocates giving these drugs in the largest dose possible, and for a long period of time.

TIC

(Habit Chorea; Habit Spasm; Motor Tic; Palmus)

Definition.—A tic has been defined to be[1] "a coördinated purposive act, provoked in the first instance by some external cause or by an idea; repetition leads to its becoming habitual, and, finally, to its involuntary production without cause and for no purpose, at the same time as its form, intensity,

[1] Tics and Their Treatment, Meige and Feindel.

and frequency are exaggerated; it thus assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse, its suppression associated with discomfort. The effect of distraction or volitional effort is to diminish its activity; in sleep it disappears. It occurs in predisposed individuals, who usually show other indications of mental instability."

Etiology and Symptoms.—The disease is especially apt to develop in those who possess a neuropathic ancestry, and who themselves are subject to neurasthenia, hysteria, and other psychoneuroses. Most cases are originally excited by an effort to relieve some peripheral irritation, thus a man developed a tic of the tongue by licking a sore on the lip. Tics may develop at any age and any group of muscles may be affected. We have facial or mimic tics, tics of the nose or sniffing tics, of the lips or sucking tics, of the tongue or licking tics, of the neck or nodding tics, of the trunk muscles, of the arms, of the hands or scratching tics, of the legs or leaping tics, bowing, whistling, sobbing, coughing tics. Any voluntary and purposive act may be imitated. The movements are usually quick and are exaggerations of normal acts. Tics may be acquired by imitation of one who has the disease.

Diagnosis.—It must be distinguished from a true spasm. Meige (*loc. cit.*) has defined a spasm to be the motor reaction consequent on stimulation of some point in a reflex spinal or bulbospinal arc. In other words it is due to actual irritation. The points of difference have been summed up by Patrick:[1] Tic is more common than spasm, and invariably develops in a nervous or neuropathic individual. Disposition or temperament has nothing to do with spasm.

Spasm is devoid of voluntary or involuntary control, while tic to some extent is always under the control of the will and always subject to involuntary control by strong emotional or intellectual pre-occupation. Spasm is an anatomical, tic a physiological disorder. Voluntary simulation of a true spasm is practically impossible. The patient

[1] Jour. Nerv. and Ment. Dis., January, 1909, p. 1.

can always repeat or imitate his tic movement and another person can nearly always do so. A spasm in the beginning may be confined to part of a muscle or of the muscles taking part in a movement (fascicular contractions). A tic always involves all the muscles taking part in any one physiological movement.

In chorea the movements are irregular, non-purposive, and largely involuntary, and are increased by excitement. Tic convulsif or De la Tourette's disease is tic plus mental symptoms (see below).

Prognosis and Treatment.—As regards cure the prognosis is doubtful. It may last for years and does not cause death. The treatment is educational, that is, training the patient to perform movements the opposite of those involved in the tic. This can be done before a mirror. The neurotic diathesis should be treated by fresh air, easily digested nutritious food, freedom from excitement and strain.

Tic Convulsif.—Synonyms and Etiology.—This disease is also known as Gilles de la Tourette's disease. It may begin as early as the sixth year. A neuropathic ancestry can usually be traced.

Symptoms.—The symptoms consist of: (*a*) *Muscular movements,* like those of simple tic (p. 430). The muscles of the face are most commonly affected, the muscles of the entire body may be.

(*b*) *Involuntary cries* and *explosive utterances.* Sometimes the words used are of a profane or obscene nature (coprolalia); in other cases there is an irresistible tendency to repeat names or sentences heard (echolalia). Movements made by other persons may also be imitated (echopraxia).

(*c*) Some cases present other remarkable mental features, which, however, may occur in neurotic subjects in whom tic is not present. They consist of *fixed ideas;* morbid impulses, etc. (p. 454). Manifestations of hysteria may also be present.

Diagnosis, Prognosis, and Treatment.—The mental symptoms distinguish the disease from simple forms of tic. The disease is usually incurable. According to Osler, recovery may rarely occur. The *treatment* consists of isolation, tonics, and antispasmodics, and educational measures. (See Tic.)

OTHER AFFECTIONS RESEMBLING CHOREA

Saltatory Spasm.—Under this title, and also as latah, myriachit, and jumpers, has been described an affection in which, only when the patient attempts to stand, there are strong contractions of the leg muscles, which cause a jumping or springing motion. It is more frequent in men, and the subjects are markedly neurotic. It may be transitory or persist for years. It sometimes occurs as an endemic neurosis, especially in Maine and Canada, there known as jumpers; in parts of Russia, known as myriachit; and Java, known as latah. Echolalia and echokinesia are often present in these cases.

Paramyoclonus Multiplex.—Synonyms and Definition.—This disease, also known as myoclonus multiplex, convulsive tremor, myospasm, myokimia disease (Dana), and fibrillary myoclonia, is rare. It is characterized by quick, clonic, spasmodic contractions of the muscles of the trunk and extremities, occurring usually in neurasthenic or hysterical subjects.

Etiology.—Fright, shock, and mental strain may excite attacks. Tuberculosis may be a factor.

Symptoms.—The symptoms are bilateral spasms, involving the same muscles on both sides and occurring only at intervals; as much as a week may intervene. The muscles most involved are those of the trunk and proximal parts of the limbs. A peculiarity of these contractions is that while they are severe no, or comparatively little, movement of the limb is produced. Another is that muscles which are incapable of being voluntarily contracted alone, such as the sartorius and supinator longus, during the attack may be seen to contract independently of other muscles. The diaphragm is also sometimes affected. The tendon and skin reflexes are increased. There are no disturbances of sensation and no mental disorder. The spasms begin usually in the shoulder and arms, and are rapid and violent, varying from 5 to 180 per minute. Tapping the tendons or irritating the skin may bring on an attack; emotional excitement may also

have a similar effect. The *pathology* of this disease is unknown. Friedreich, who first described it, believed that it was due to an irritation of the anterior horn cells. In a case studied by Hunt[1] nothing was found excepting hypertrophy of the muscle fibers.

Diagnosis.—It must be distinguished from hysterical chorea, which is possibly a form of myoclonus, hereditary chorea, and tic. The peculiarities of these disorders will be found on pages 428, 429, and 430, when the differences will readily be seen. Related to this disorder is a fibrillary twitching of the muscles known as myokymia or fibrillary myoclonus of King. Family types of myoclonus also exist, first described by Unverricht, which are possibly related to hereditary chorea, but the movements are much more severe. This form of myoclonus may also be associated with epilepsy[2] (p. 440). Muscular spasms may also be caused by exposure to intense heat (p. 440).

Prognosis.—The prognosis is usually unfavorable. Often it lasts for years.

Treatment.—Treatment consists of antispasmodics, attention to the general health, and the administration of tonics, such as iron and arsenic, static electricity (spark or breeze), and galvanism, applied by passing strong currents down the spine.

Posthemiplegic chorea and **athetoid movements** are described on page 37, and **hysterical chorea** on page 428.

DYSBASIA LORDOTICA PROGRESSIVA

This affection, also termed *dystonia musculorum deformans* and *tortipelvis*, has recently been described by Oppenheim.[3] It occurs in children and young adults of the Jewish race, and consists of a deformity around the pelvis and of tonic and clonic myospasms of the musculature around the pelvic girdle, associated or not with similar twitchings of other

[1] Jour. Nerv. and Ment. Dis., June, 1903, p. 408.
[2] See article of Hunt quoted above.
[3] Fraenkel, Jour. Nerv. and Ment. Dis., June, 1912, p. 361.

muscles. In a reclining position symptoms are not present, excepting some deformity about the pelvis. Attempts to stand and especially to walk bring on the spasms. The gait is peculiar, having been termed a "monkey or dromedary gait." There is marked lordosis of the lower dorsal and upper lumbar parts of the vertebral column and prominence of the buttocks. Symptoms of organic disease of the nervous system are absent.

EPILEPSY

Definition and Varieties.—An affection of the brain characterized usually by paroxysmal attacks of unconsciousness, with or without convulsions, more rarely by convulsions without loss of consciousness. The loss of consciousness with convulsive seizure is known as *grand mal;* transient loss of consciousness without convulsion as *petit mal;* localized convulsions without loss of consciousness are sometimes the manifestation of *Jacksonian epilepsy.*

Etiology.—A large proportion of the cases begin before the age of puberty. Of 1450 cases analyzed by Gowers, 422 developed the disease before the tenth year, and 75 per cent. began before the twentieth year. Most of the cases are of the so-called *idiopathic* form, in which no cause can be determined. Others are due to organic brain disease, especially causes producing cerebral palsies of children (p. 249). Patients developing the idiopathic form often are children of neurotic families, those in whom neuralgia, hysteria, and insanity prevail. Chronic alcoholism and syphilis, particularly the former, in the parents may also act as predisposing causes. Rachitic children are probably also predisposed. Trauma, exclusive of that causing visible brain injury, some reflex irritation, as worms, teething, indigestible food, adherent prepuce, or nasal disease, may act as exciting causes in those predisposed. Infectious fevers may also act in the same way.

Epileptiform convulsions developing in persons past thirty-five are rarely idiopathic, and may be due to one

of the following causes: *Organic brain disease*, as either tumor, abscess, syphilis, traumatism causing fracture of the skull, or the result of a previous apoplectic attack.

Toxemias, notably alcohol, lead, uremia, diabetes, syphilis (when a parasyphilitic disorder), products of intestinal putrefaction—this, while rarely an exciting cause, certainly aggravates the symptoms in those who already have the disease. Meyers[1] has called attention to the occasional association of epileptiform convulsions and symptoms of hyperthyroidism. The author has seen a case in which convulsions followed the taking of large doses of thyroid extract to reduce flesh.

Reflex Irritation.—True reflex epilepsy is rare, but eyestrain, nasal and pharyngeal disease, as adenoids or some other source or irritation, may now and then be the cause of epileptiform convulsions.

Circulatory disturbances, due to weak heart from any cause; arteriosclerosis, Raynaud's disease. Epileptiform seizures occur as one of the manifestations of Stokes-Adams disease. In this they are associated with a very slow pulse.

Epileptiform convulsions may also occur in the course of *multiple sclerosis*, *paresis*, and *dementia præcox*.

Morbid Anatomy and Pathology.—No constant changes have yet been discovered in the brain of epileptics. Degeneration of Ammon's horn has, however, been found in a number of cases (60 per cent., Alzheimer), and various forms of organic brain disease (see Etiology) may cause epileptiform convulsions. Changes in all of the ductless glands have been reported.

Symptoms.—(1) Grand Mal.—*Precursory symptoms*, consisting of one or more of the following, viz., irritability of temper, mental changes amounting to insanity, persistent giddiness, headache, sudden jerkings, voracious appetite, may in rare instances precede, for several days, the fit.

In about one-half the cases a peculiar sensation of movement in some part of the body directly precedes the convulsion; this is known as the *aura*. It may consist of:

[1] Monthly Cycloped. and Med. Bull., May, 1912.

(*a*) Either a motion or sensation, or both, in some part of one-half the body, usually the hand; if a sensation, it is a numbness or tingling; if a motion, it is usually twitching of the fingers. It may then ascend the arm to the head, trunk, and then involve the leg before consciousness is lost. It may begin in the leg, face, or tongue, and may pass down instead of up the limb. This form of aura is more frequent in cases of focal brain lesions.

(*b*) Sensations of motion in both limbs; general tremor or shivering; sense of general malaise; of heat or a feeling of faintness.

(*c*) Visceral or pneumogastric warnings, consisting of vague sensations or pain in the epigastrium, sometimes associated with nausea; peculiar sensations ascending from the epigastrium to the throat or head, sometimes producing a sense of choking; cardiac palpitation or pain in that region.

(*d*) Cephalic sensations, consisting of giddiness, subjective or objective; turning of the head and eyes; headache; somnolence.

(*e*) Psychical auræ, consisting of a certain emotion or idea, always the same in the same case; a feeling as if something were wrong.

(*f*) Special sense auræ are frequent. If olfactory sensations, they are usually an unpleasant smell. These are sometimes due to organic brain disease involving the uncinate gyrus.[1] Gustatory sensations, consisting of sour, bitter, or metallic taste; auditory, as sudden loss of hearing, peculiar sounds. Visual auræ are the most common; these may be either flashes of light or hallucinations (p. 349).

(*g*) Rarely the attack is preceded by some coördinated movement, as running for some distance (procursive epilepsy), or whirling about.

Either a loud cry or peculiar groan sometimes is given just at the *onset of the attack*. As the *convulsion begins* the patient falls, tonic spasm causing the head to be rotated or drawn backward, and sometimes deviation of the eyes and

[1] Hughlings Jackson has described peculiar attacks that he terms uncinate fits. They consist of taste aura, chewing movements, and a peculiar dreamy state. Brain, 1898, p. 580. Organic lesions of the uncinate convolution may cause such symptoms.

mouth to the side most affected sets in. The hands are clenched, the thumb often being inside the fingers, and the arms flexed at the elbows and the hands at the wrist, and the legs extended. Owing to the impeding of the respiration the initial pallor gives way to lividity. The pupils are usually dilated and insensitive to light. The tonic spasm lasts but a short time, and gives way to clonic spasm, in which all the muscles are in violent contraction, saliva exudes from the mouth, and, if the tongue is bitten, as it usually is, is blood-stained. Urine and feces may be discharged, and the cyanosis gradually lessens. This stage usually lasts two or three minutes. After the contractions lessen the patient remains comatose, the face congested, and the breathing stertorous. In a short while he can be aroused; but if left to himself, sleeps for some hours. Afterward he may complain of headache or mental confusion.

In rare instances the patient may remain in a condition of *trance*, in which he may perform apparently purposive and logical acts; he may go on journeys, and conduct conversations, etc., of which he has no recollection afterward. Crimes have been committed while in this state. This is known as *epileptic automatism*, an epileptic equivalent, and may occur without the preceding convulsion. He may become maniacal and homicidal. Such attacks may also occur in place of the fit. (See Epileptic Insanity.) Patients who have frequent attacks become demented sooner or later.

Transient paralysis, often hemiplegic in type, or aphasia, may follow an attack. Very rarely such paralysis may occur in place of the convulsion. Attacks may occur *at night* during sleep; in some patients they only occur then. Occasional incontinence of urine at night, waking in the morning with headache, and mental confusion are suggestive of nocturnal epilepsy.

If a number of attacks follow each other in rapid succession, the condition is termed the *status epilepticus*. In this the temperature may rise, and after the convulsions cease the patient becomes greatly prostrated and delirious.

(2) Petit Mal. — These attacks consist of transient unconsciousness, sometimes accompanied by a feeling of

giddiness or faintness. They are of very brief duration. Usually the patient will be noticed suddenly to stop whatever he is doing. In some cases the eyes become fixed and the face slightly pale; in others there is no change of color. If anything is in the hand at the time, it is usually dropped. In a second or two the attack passes off and the patient resumes what he had been doing, as if nothing had happened. The attack may be preceded by a warning sensation, and in many patients this is the only thing which enables them to know that they have had a seizure, as the period of unconsciousness is so momentary that they are unaware of it (psychic epilepsy). Gowers mentions the following as the chief characteristics of petit mal, arranged in the order of frequency:

1. Sudden momentary unconsciousness, or "fainting," or "sleepiness," without warning.
2. Giddiness.
3. Jerks or starts of the limbs, trunk, or head.
4. Visual sensations or loss.
5. Mental state: sudden sense of fear, etc.
6. Unilateral peripheral sensation or spasm.
7. Epigastric sensation.
8. Sudden tremor.
9. Sensations in both hands.
10. Pain or other sensation in the head.
11. Choking sensation in the throat.
12. Sudden scream.
13. Olfactory sensation.
14. Cardiac sensation.
15. Sensation in nose or eyeball.
16. Sudden dyspnea.
17. General "indescribable" sensations.

After these attacks patients may perform automatic actions, as after attacks of grand mal. Convulsions usually sooner or later develop, and the two forms often coexist.

Jacksonian Epilepsy.—These attacks are usually the result of irritative lesions affecting the motor region of the cortex. Attacks of this nature have also been caused by growths in other parts of the brain, as in the cerebellum and

pons, and in acquired hydrocephalus. In such cases the symptom appears some time after other signs of disease have been present. Therefore, such convulsions, when occurring for the first time, long after the general signs of intracranial growth have appeared, are to be disregarded as localizing signs.[1] They also may be caused by either uremia or paretic dementia, and sometimes occur as a manifestation of idiopathic epilepsy. They are peculiar in that consciousness is either not lost at all or is lost late. In a typical attack there is usually a feeling of tingling or numbness, succeeded by spasm in a limited group of muscles of the face, arm, or leg. For instance, the mouth may be drawn to one side or a finger twitch. The muscles first affected indicate the seat of lesion. The spasm then spreads to adjacent centres and gradually involves all the muscles of the limb or face, without loss of consciousness. In more severe attacks the spasm finally involves the entire side, and then becomes general, with loss of consciousness. A condition which might be confounded with Jacksonian epilepsy occurs sometimes in those who have been exposed to long-continued intense heat. It consists of violent tonic spasms occurring at intervals during a period of several hours in the muscles of the legs, forearms, and abdomen. Fibrillary tremors in the calf muscles are also present. The facts that the spasms are very painful and may be excited by voluntary motion as well as the history should prevent error.[2]

Myoclonus epilepsy is characterized by epileptic seizures of the ordinary type, and more or less constant myoclonic spasms, varying from fibrillary tremors (myokimia) to violent spasms of the large muscles, sufficiently intense to throw the patient down. It may possibly be due to a poisoning by parathyroid secretion.[3] It may be a family disease.

Diagnosis.—Attacks of petit mal may have to be distinguished from syncope and vertigo. From syncope the distinction rests upon the absence of obvious cause, the suddenness of the onset and return to the normal condi-

[1] Collier, Brain, 1904, p. 498.
[2] Edsall, Jour. Amer. Med. Assoc., December 5, 1908, p. 1969.
[3] Clark, Review Neurol. and Psychiatry, July, 1907, p. 532.

tion, the absence of weakness of the pulse, and a history of frequent previous attacks. Vertigo is rarely attended by unconsciousness; the giddiness lasts longer, and the return to normal is more gradual. In Ménière's disease evidence of disease of the internal ear will be present. (See Aural Vertigo.)

When the attack consists of convulsive seizures, it is necessary to distinguish them from those due to hysteria. The following, from Gowers, illustrates the differences between them:

	Epilepsy.	Hysteria.
Apparent cause	None.	Emotion.
Warning	Any, but especially unilateral or epigastric auræ.	Palpitation, malaise, choking, bilateral foot auræ.
Onset	Always sudden.	Often gradual.
Scream	At onset.	During course.
Convulsion	Rigidity, followed by jerking, rarely rigidity alone.	Rigidity or "struggling," throwing about of limbs or head, arching of the back.
Biting	Tongue.	Lips, hands, other people or things.
Micturition	Frequent.	Never.
Defecation	Occasional.	Never.
Talking	Never.	Frequent.
Duration	A few minutes.	More than ten minutes, often longer.
Restraint necessary	To prevent accident.	To control violence.
Termination	Spontaneous.	Spontaneous or induced (water, etc.).

Also in the epileptic convulsion there is absence of the pupillary reflex; in hysteria this is usually present. The initial pallor which precedes the facial lividity is peculiar to epilepsy. As this period is very brief, close observation may be required in order to detect it.

It should be remembered that hysteria may occur in epileptics.

Having determined that the attacks are epileptic, it is necessary to determine whether they are idiopathic or due to some of the ascertainable causes mentioned. Attacks occurring for the first time in a person over thirty-five years are usually not idiopathic, but may be rarely. In this connection it must be remembered that long remissions may occur, and careful inquiry must be made to be sure that the

attack is actually the first. If it is so, then careful search must be made for the existence of the causes mentioned.

In Jacksonian epilepsy the cause, whether tumor, lesions causing infantile cerebral palsies, localized meningo-encephalitis, abscess, uremia, or paretic dementia, must be determined.

Prognosis.—Death rarely occurs during a fit, unless the patient is injured by the fall or asphyxiated, which latter may sometimes happen in nocturnal attacks if the face becomes buried in the pillow. Sudden death may also rarely occur from acute pulmonary edema.[1] The status epilepticus often causes death by exhaustion.

It is probable that a small percentage of cases recover, and this result is most likely to be attained in those cases in which the fits are not frequent and active treatment is commenced early and continued. But it is impossible to predict recovery in any case. It must also be remembered that long remissions (twenty-nine years in a case mentioned by Sinkler) may take place.

In organic epilepsy, if due to syphilis, the prognosis is fair. When due to either tumor, or a postapoplectic cyst (p. 324), or a depressed fracture, a certain number may be cured if operated upon early. If the spells have lasted two years the outlook as regards cure of the fits is not so good.[2]

Treatment.—A child who develops convulsions from any cause should be treated carefully for some time after, for in a predisposed child a convulsion due to some peripheral irritation may be the starting point of future epilepsy. Hence all sources of irritation should be removed, the child put under the best possible hygienic conditions, and small continued doses of antispasmodics given. During an epileptic convulsion no special treatment is necessary; the clothing may be loosened, and something placed between the teeth. Epileptics should have an easily digested diet, meat

[1] Ohlmacher, Amer. Jour. Med. Sci., March, 1910, p. 417.

[2] When either a tumor or postapoplectic cyst is the probable cause, the only cases proper for operation, at least when undertaken for the cure of the seizures, are those in whom the attacks are of the Jacksonian type. Tumors may be operated on for other reasons (p. 363).

being allowed but once a day. Overloading of the stomach should be avoided. Small berries with hard seeds, cake and pastry in any form, pork, veal, ham, or anything fried in grease, all alcoholic drinks, are especially to be abstained from. Some fixed employment is beneficial. Excitement of any kind should be avoided.

Colonization of a large number of epileptics, where they can have the benefit of country air, outdoor employment, and education, has proved of much value. If the aura precede the attack sufficiently long, it may sometimes be aborted by the inhalation of nitrite of amyl.

To influence the seizures the bromides, preferably ammonium or strontium, are the most useful; their effects are usually increased by the addition of antipyrine, as recommended by H. C. Wood. For an adult, 10 to 30 grains of the bromides and 5 or 6 grains of antipyrine may be given three times daily, according to the effect desired. Care must be taken not to produce bromism, and only sufficient of the drug given to control the paroxysms. Atropine, alone or combined with a bromine salt, may also prove of service. Some cases do not appear to be benefited by the bromides; in fact, the symptoms may be aggravated; in such, or when decided symptoms of bromism occur, the fluidextract of Solanum carolinense, in doses of a teaspoonful or more, t. i. d. for an adult, may be tried. Good results have been attained in some cases from the use of thyroid extract. Peterson obtained improvement by its use for the mental symptoms in cases where dementia was marked. Gordon also states that it is beneficial in those cases where the seizures only occur about the menstrual period. He gives it between the periods and bromides for a few days before the expected attack. If the heart is weak, cardiac tonics (except strychnine) should be given. Nitroglycerin may be of service, especially if there is arteriosclerosis. In addition to these measures, the general health must be maintained by arsenic and other tonics, and the digestive organs kept in good condition by means of laxatives and intestinal antiseptics, and all sources of peripheral irritation removed. In Jacksonian epilepsy the question of the advisability of a

surgical operation may be raised. This is justifiable under the following conditions, viz.:[1] If not more than two years have elapsed since the traumatism or beginning of the disease which causes the seizures. If there is a depression of the skull, operation is warranted at any period. In focal epilepsy without visible lesion excision of the centres is justifiable if the symptoms have lasted but a short time. But one must not be too sanguine of obtaining a cure, as the very scar caused by the excision may in itself cause epileptic seizures. This has occurred after the removal of a tumor. Of course, if there is reason to suspect a growth in the region accessible to operation it is always justified. (See Brain Tumor.) When the status epilepticus occurs, chloral and hypodermic injections of either morphine or hyoscine should be tried, and the bowels washed out with either high enemas or purgatives. Amylene hydrate in doses of 3 grams per rectum and repeated if necessary has been recommended. Chloretone in 5-grain doses three times daily may also be of service. In Jacksonian epilepsy a tight band applied about the limb when a seizure is felt approaching, will often arrest it.

NEURASTHENIA

Synonyms and Definition.—This affection is commonly known among the laity as either nervous prostration, nervous debility, or nervous exhaustion. It is a condition in which there is more or less marked and persistent diminution of nervous energy, together with an increased reaction, mental and physical, to external impressions. In other words we have two principal symptoms: nervous weakness and nervous irritability (Dercum).

Etiology.—Neurasthenic parents, or those who are alcoholic, tuberculous, or syphilitic, are predisposing causes. Children begotten by parents who are old are also apt to become neurasthenic. The overworking, either mental or physical, of children may lay the foundation of future neurasthenia.

[1] Condensed from conclusions reached by Sachs and Gerster, Amer. Jour. Med. Sci., vol. cxii, p. 39.

The exciting causes are overwork, either mental or physical; alcoholic and sexual excesses, prolonged physical or mental strain and occupations which entail these, excessive child-bearing and prolonged lactation, infectious diseases, especially influenza; physical injury and emotional shock (p. 478); excessive use of tea, coffee, and tobacco; autointoxication due to the absorption of the products of intestinal putrefaction, as would occur in chronic constipation and gastro-intestinal indigestion. It may also accompany or follow other chronic and exhausting diseases, as tuberculosis, anemia, chlorosis, diseases of the sexual organs, especially chronic prostatitis, syphilis, arteriosclerosis, ptoses of various organs, pellagra[1] especially in the early stages, and be associated with hypochondriasis and hysteria.

Pathology.—In the light of our present knowledge, neurasthenia is a functional disease. C. F. Hodge has shown that in cells fatigued by constant work the nucleus decreases in size, becomes irregular in outline, loses its reticulated appearance, stains darker, and that the cell protoplasm shrinks slightly in size and stains more feebly. It is possible that in persons who for a long time overwork their nerve cells or expose them to any of the influences mentioned under etiology they lose their power of recovery, and these changes remain permanent.

Symptoms.—Patients with neurasthenia describe their **condition** as follows: They "are nervous," they get easily excited and irritated, they cannot do as much physical work as they formerly did, and in attempting any mental effort they become confused and are unable to concentrate their attention. They will also complain of insomnia, depression of spirits, indigestion, and palpitation of the heart. In some, most of the complaint is about the sexual organs, of excessive nightly emissions, loss of sexual power, etc. When carefully examined, such patients will be found to present motor, sensory, psychic, and somatic symptoms.

Motor Symptoms.—In the majority of cases there will be general muscular weakness; he cannot walk as far as formerly

[1] Tucker, Amer. Jour. Med. Sci., March, 1912, p. 332.

in fact, tires sooner after any physical exertion. For a few moments he may be able to exert his usual strength, but it soon becomes exhausted. A common symptom is fine tremor, generally but not always limited to the hands and tongue, and increased by exertion. Fibrillary muscular tremors and spasms of the muscles may also be observed. The *knee-jerks* are usually increased, but this differs from that due to organic cause in the fact that, if tested repeatedly, they soon become exhausted and hence diminished. Instead of this, they may be normal, lessened, or absent, in which case they can be elicited by reinforcement (p. 62). The tendon reactions in the arms may also be increased. The tendon reactions may vary, being present at one time and absent at another.

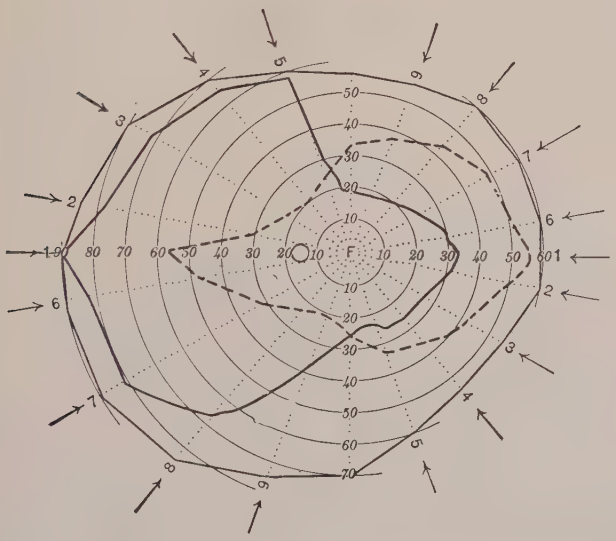
Sensory Symptoms.—These may consist of a feeling of malaise and a constant feeling of tire and exhaustion. A feeling of tightness about, or pressure upon, various parts of the body often exists. More or less pronounced vertigo is a frequent symptom. It is usually paroxysmal, following exertion, but may be constant. Dull pains and aches in various parts of the body, especially the head, back, and legs, are complained of. With the headache of neurasthenia there is often a feeling of constriction, as if a tight band were about the head, or there is a sense of pressure upon the top of the head.

Backache is usually referred to the small of the back, although often there may be burning sensations in the scapular region. Along with the pain there are often found areas of great tenderness along the spine. They are elicited by light pressure; may change their location from time to time, but are most commonly located over the seventh cervical vertebra, the middorsal region, the dorsolumbar junction, the midlumbar region, and over the sacrum and coccyx. These areas may exist without spontaneous pain being present. This symptom has been sometimes incorrectly termed "spinal irritation."

There may be cutaneous hyperesthesia in various regions and paresthetic sensations, as numbness, tinglings, sensations of heat or cold, etc

The *special senses* may also suffer; the *eyes* soon tire, brief use for reading or other fine work causing blurring, confusion, headache, and vertigo. To attribute these symptoms to neurasthenia, however, refractive errors must be excluded. The visual field may be slightly contracted. More rarely there is retinal hyperesthesia. A symptom sometimes seen is inability to completely close the eyes when standing with the feet close together; associated with

Fig. 116

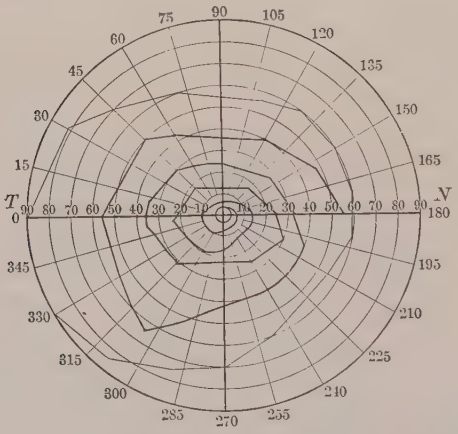


Förster's shifting or displacement type of the visual field. (Wilbrand.)

this is fibrillary twitching of the orbicularis (Rosenbach's sign). Some neurasthenics, especially women, complain that on awakening in the morning they find it impossible to raise the eyelids without assistance from their fingers (night ptosis of Gowers); it may also occur if they awaken during the night (night ptosis of Weir Mitchell). This symptom also occurs in hysteria. Changes in the visual fields are important symptoms, although they may be found in other conditions, notably hysteria (p. 466). Those occurring in

neurasthenia have been termed "fatigue contractions." This contraction may be very marked; it may appear in all of the four halves of the visual fields, or only in certain ones (Fig. 116). The field may be normal when first tested, but owing to fatigue, contractions in different meridians soon occur. The contraction is not permanent until recovery ensues, as it is apt to be in hysteria and changes in color sequence do not occur.[1] Another form is the fatigue or exhaustion spiral field, in which the points when united form a tracing like a watch spring (Fig. 117). Patients may also complain of objects appearing misty, or of specks floating in front of the eyes (muscæ volitantes).

Fig. 117



Fatigue spiral field. (Von Reuss.)

Various forms of *tinnitus aurium* are often present and hyperesthesia of the auditory nerve may be the means of causing patients to suffer much from slight noises.

Taste may be impaired or disagreeable tastes may be complained of.

Psychic Symptoms.—The ability to sustain prolonged intellectual effort is interfered with. If attempted, in a short

[1] De Schweinitz, The Eye and Nervous System, edited by Posey and Spiller, p. 671 et seq.

time confusion, lack of concentration, headache, and vertigo appear. Such patients will complain that they are "losing their mind" or "their memory." Investigation will show, however, that their memory is good. There is disinclination to think or try to perform mental labor, and self-confidence is lost. There is usually marked irritability of temper, and often loss of affection for those formerly held dear. The patient may also be excessively emotional.

Neurasthenics become introspective; they are continually watching for new symptoms and dwelling upon and consequently unconsciously exaggerating the old, and attaching undue importance to them.

Morbid fears, anxiety, and obsessions may be suffered from, but these are more frequent in psychasthenia (p. 454). Insomnia is an early and frequent symptom; the sleep may be restless or the patient falls asleep normally, but awakens much sooner than he should. Sleep is not refreshing, and the patient complains of feeling more tired in the morning than when he retired. Horrible dreams may be complained of.

Somatic Symptoms.—These consist of disturbances of digestion, of circulation, of secretion, and of the sexual functions. *Digestive disturbances* are very common; while, as has been mentioned under Etiology, they may cause neurasthenia, it is also true that in some cases they are caused by it. They are usually manifested by feelings of heaviness and discomfort in the epigastrium, appearing a great or less length of time after eating. In more aggravated cases, acid eructations (heartburn), flatulence, nausea, and even vomiting may be present. The tongue may be coated, but more commonly is clean, but flabby and marked with the teeth. Constipation is the rule. In some cases the appetite is increased, but food does not satisfy hunger.

The most common *circulatory disturbance* is palpitation of the heart. It is often associated with the gastro-intestinal disturbances mentioned above. There may be sensations of pain in the cardiac region during these attacks, and they may resemble angina pectoris. Instead of palpitation, the pulse rate may be permanently increased. Vasomotor symptoms are common. A frequent one is aortic pulsation, caus-

ing a deep-seated throbbing in the epigastrium. It may be often felt by the hand. Flushings of the face and other parts, and coldness and lividity of the extremities are also frequent.

There may be *lessening of all the secretions*—viz., urine, saliva, perspirations, etc.—or they may be *increased*, especially the perspiration. This increase may be only noticed at nights, when night sweats occur. Even when the actual quantity of urine is not increased, frequent urination is a common symptom. The urine often contains an excess of uric acid and urates, of phosphates, or of oxalates. Phosphatic urine is cloudy while being passed, and such patients often imagine that they have spermatorrhea.

Sexual.—In some cases these may dominate the picture of the disease, such cases being often termed sexual neurasthenia. In others they are secondary to other symptoms. In men they consist of frequent nightly emissions, of premature ejaculation when sexual intercourse is attempted, incomplete erections, absence of pleasurable sensation, and various paresthesias referred to the genital organs. Spermatorrhea is imagined, especially if the urine is phosphatic or there is a discharge of prostatic secretion, which often occurs. Women may also suffer from involuntary orgasms at night and sexual weakness. They are apt to have pains in the pelvic region, and, possibly, ovarian tenderness *without* the existence of *organic disease of the organs*. It must be understood that all of the symptoms detailed do not occur in every case. Also, that while neurasthenia is a disease of the general nervous system, one part may be affected more than another; in one, cord symptoms predominate; in another, cerebral.

Diagnosis.—It is important to remember that in rare instances cerebral growths, especially those involving the frontal lobes, may for a long time present none but symptoms resembling those of neurasthenia. Also that grave organic diseases of other organs may so begin—viz., tuberculosis, pellagra, diseases of the blood, gastric cancer, dilated stomach, gastroptosis, chronic uterine or ovarian disease (see above). Careful examination should be made to detect the presence

of any of these disorders. Primary neurasthenia must be differentiated from hysteria, hypochondria, exophthalmic goitre, chronic alcoholism, melancholia, and the commencement of paretic dementia.

Hysteria major is distinguished by convulsions, areas of anesthesia, reversal of the color fields, various forms of paralysis, and contractures. In hysteria of a milder type these may be absent, but the onset of the symptoms is apt to be sudden; they vary from day to day, being caused by suggestion and often removed by the same means (p. 455); insomnia is not common; headache, if present, is of the peculiar type known as hysterical clavus; globus hystericus will probably be complained of; there may be intervals in which the health is good. It must be remembered the neurasthenia may be a predisposing cause of hysteria, hence symptoms of the two often coexist. The following table by Ziehen may prove of service.

Hysteria	Neurasthenia
Mostly unilateral; patch-like or regional anesthesia, hypesthesia and hyperesthesia, and analgesia, hypalgesia and hyperalgesia.	Sensibility intact or generally increased.
Pressure points with hysterogenic and eventually also hysterophrenic characters more marked mostly in one side, usually combined with hyperesthesia.	Pressure points without such characters, usually symmetrical and with intact skin-sensibility.
Paralyses not unusual. Headache localized in spots, rarely a sense of pressure.	Nearly always abnormal tire. Headache often band-like and sense of pressure frequent.
Visual fields diminished.	Visual fields only diminished under the influence of fatigue.
Smell, taste, and hearing often involved.	Smell, taste, and hearing involved symmetrically and usually in the sense of a hyperesthesia.
Skin reflexes often unequally modified.	Skin reflexes rarely different on two sides of body.
Often typical attacks.	No attacks—or rarely attack-like emotional movements set up by affect action, mostly depressive ideas.
Mood excessively variable.	Mood irritable or hypochondriacal.
Intellectual activity disturbed, especially the attention.	Intellectual activity disturbed by reason of early fatigue.
Marked suggestibility.	Suggestion influences slight.
Sleep often excellent.	Sleep mostly bad.
Course polymorphous.	Course rarely polymorphous.

Although *hypochondriacs* may complain of various symptoms, examination will show that the organs complained of are in good condition, and that their functions are performed properly. Here, again, however, a neurasthenic may become hypochondriacal.

The presence of exophthalmos, enlarged thyroid, and extreme tachycardia distinguishes *exophthalmic goitre*. In cases where these symptoms are not marked, the diagnosis may be doubtful.

The early stages of *melancholia* may simulate neurasthenia, but differs in the more marked depression, more marked insomnia, refusal to eat, and possibly the presence of delusions and suicidal impulses.

The early stages of *paretic dementia* are also characterized by neurasthenic symptoms, but the loss of memory, tremor of the facial muscles and tongue, unequal and Argyll-Robertson pupils and a feeling of well-being, will distinguish that disease. *Dementia præcox* especially in the early stage (p. 550) may be mistaken for neurasthenia.

Prognosis.—Neurasthenia is a chronic disease, and may last for years with intervals of improvement. Recovery may occur, but any undue strain is apt to produce a relapse.

Treatment.—Cases of primary neurasthenia are best treated by the so-called "Rest Treatment" (p. 93). If the case is mild this need not be carried out absolutely, but can be modified so that the hours of rest are prolonged while those of work are curtailed. If the symptoms are principally cerebral, motor weakness not being a factor, an outdoor life, such as can be secured from a gunning trip, or a sea voyage, or a trip to the seashore, will often cause the desired result. Neurasthenic patients do better in a mild climate. Or, if this cannot be obtained a certain amount of regulated outdoor exercise, as bicycle or horseback-riding, or golf, with lengthened hours of rest and regulation of diet, may be sufficient. The regulated employment of the patient in certain forms of manual labor, as basket-making, metal-working, wood-carving, etc., has given good results,[1] as has

[1] Hall, Jour. Amer. Med. Assoc., July 23, 1910, p. 295.

also camp life and a systematic course of exercises and feeding.[1] Cold douches to the spine, followed by vigorous friction with a coarse towel, is an excellent tonic; but care must be taken not to begin with too low a temperature. If desired the cold may be alternated with a hot one, beginning at 90°, running up to 105°, and then down to 70°, the treatment lasting about four minutes. Salt rubs are also of benefit. Warm baths (95° to 100°) at night, or wet packs (p. 92) are often useful to promote sleep and relieve irritability and restlessness. It is well also to advise the use of considerable quantities of pure water internally. The bromides may be used to allay the irritability, but should not be used continuously. The writer has gotten good results from a combination of bromide and arsenic. Antipyrine or phenacetin are also useful for this purpose, as is also valerian. The condition of the alimentary canal must be looked after. A diet of milk, eggs, fish, green vegetables, and fruits, and but little meat, being most suitable. Constipation must be relieved. Intestinal antiseptics are often useful. Massage gives good results, even when the "rest cure" is not being undergone; it improves the condition of the digestive organs and circulation, and promotes sleep. Electricity in the form of general faradization, static insulation, or high-frequency currents may also be of service.

Of drugs to build up the nervous system, nux vomica, mineral acids, arsenic, iron, phosphorus, or the glycerophosphates are all of service. Much strychnine, and in some cases all, is to be avoided, as it often increases the irritability. Insomnia should be treated according to the rules detailed on page 115. The headache is usually benefited by the static breeze, massage of the head, or the application of cold. The tenderness of the spine should be treated, if it is excessive, with either small blisters, the constant galvanic current, static breeze, or high-frequency currents. The use of alcohol, tea, coffee, and tobacco must be prohibited or restricted. The use of the drugs mentioned above may be combined with the rest treatment;

[1] Orbison, Jour. Amer. Med. Assoc., January 13, 1912, p. 86.

but as the plan is expensive it is not available for many, and reliance must be placed upon hydrotherapy, hygienic measures, and drugs. Whatever form of treatment is employed continual encouragement of the patient is essential.

For the symptoms referred to the male sexual organs the use of large-sized cold sounds, combined with measures described above, is often useful. Electricity in the form of galvanism, the cathode being placed successively for five minutes each over the groins and perineum, the anode being on the spine, often gives good results. In aggravated cases the local application of silver nitrate to the prostatic urethra may be required. These cases are often best treated by a genito-urinary surgeon.

PSYCHASTHENIA

Definition and Symptoms. — When with the physical symptoms of neurasthenia, the following mental symptoms coexist. Janet[1] proposed the name psychasthenia: Obsessions (p. 525), morbid fears, anxiety (p. 455); doubts, enfeebled will power, tics (p. 430), and uncontrollable movements, sometimes of an epileptiform nature. Some of these are fear of open places (agorophobia), fear of closed places (claustrophobia), fear of being alone (monophobia), fear of crowds, abnormal fear of storms, fear of personal defilement (mysophobia), morbid dread of impending danger either to one's relative or self; doubting mania in which the patient is never sure that he has performed any action correctly, irresistible tendencies to repeat continually certain words (onomatomania), to count a certain number of times before performing an action (arithmomania), an irresistible desire to touch certain things (desire du touchee), etc. The patient is conscious of the absurdity of these actions and feelings. This fact is sufficient to distinguish the psychosis from hysteria in which it is necessary to point out to the patient the absurdity of his actions.[2]

[1] Jour. Amer. Med. Assoc., February 29, 1908, p. 665.
[2] Collins, New York Med Jour., February 15, 1908.

Treatment.—Is that of neurasthenia, especially outdoor life as the training-camp method. Psychoanalysis (p. 477) may be of service in some cases.

ANXIETY NEUROSIS
(Angst Neurosis)

Definition and Symptoms.—This is an intense anxiety or fear that evil of some nature is going to occur to self or friends. While there may be a real ground for the anxiety it is out of all proportion to the seriousness of the cause. It is a relatively excessive fear. This psychosis may occur either as a symptom of melancholia, dementia præcox, neurasthenia, or psychasthenia, but Freud has claimed that it also occurs in pure form. The mental symptoms consist of a mixture of dread, panic, terror, anguish, and apprehension, a sense of something impending, expectation of something awful or harmful. The physical symptoms may be pseudo-angina, sense of oppression, cessation of salivary and gastric flow, increase of urine and sweat, diarrhea, strangury, and paresthesias.[1]

Treatment.—The treatment is that of psychasthenia.

HYSTERIA

Definition.—To give an adequate definition of hysteria is difficult, as many different views have been advanced as to its nature. It may be truly termed a mental disturbance or psychoneurosis. The old definition of Moebius largely conforms with modern ideas: "A state in which ideas control the body and produce morbid changes in its functions." Crocq[2] more recently has stated that it is "a psychopathological state characterized by hyperimpression ability, diminution of cerebral control, and hypersuggestibility."

[1] Jones, Jour. of Abnormal Psychology, June and July, 1911, p. 81.
[2] Journal de Neurologie, 1907, No. 8.

The latter condition mentioned in this definition is a large factor in the production of many hysterical symptoms and must be remembered in examining these patients.

The views of Breuer and Freud now attracting much attention, but not adopted by many may be briefly mentioned.[1] They believe that hysteria is always due to a physical or psychical trauma which may have occurred some time before the symptoms develop. Freud believes that this trauma is practically always sexual, but not necessarily sensual, in nature. They usually have occurred before the age of puberty, and, as a rule, do not give rise to symptoms until after that period. They are due to the repression or burial of these episodes in the subconscious and hence forgotten by the conscious mind. As Freud says: "Psychoanalyses of hysterical individuals show that the malady is the result of the conflict between the libido and the sexual repression, and that their symptoms have the value of a compromise between both psychic streams." The motive for this repression is usually shame or disgust, and he claims that by ascertaining the repressed incident the symptoms will disappear (p. 477). By sexual experiences are meant those having to do with modesty, parental and filial affection, and all sexual experiences and perversions.

Etiology.—Heredity is the great predisposing cause of the disease; there is in most cases—but there are exceptions—a history of hysteria or of epilepsy, insanity, or similar neurosis in the parents.

The *exciting* causes are trauma, especially if accompanied by fright (see Traumatic Neuroses); acute and chronic disease; emotions and moral shock, as fright, disappointment, grief, and religious excitement; defective education—*i. e.*, in children the permission of habits of self-indulgence, absence of discipline, overwork at school; toxemia, thus alcohol, lead, mercury, morphine, and tobacco, may excite hysteria; and imitation (one case of severe hysteria brought in constant contact with those predisposed may excite the

[1] Brill, Selected Papers on Hysteria and other Psychoneuroses, Jour. Nerv. and Ment. Dis. Publishing Co. Also Jelliffe, Osler's Modern Medicine, vol. vii, p. 815.

disease in them). Hysteria may occur at any age, in either sex, and in every race and country. It is most common in women at about the age of twenty. It may occur in early childhood. Thus, Clopatt, in a total of 272 cases, gives 20 as occurring previous to three years of age. It is especially common among the Latin races and Jews. (See also definition.)

Symptoms.—These are divided into two classes—the *paroxysmal* and the *interparoxysmal;* the former transient, and the latter more or less permanent.

The Paroxysm.—This is known by the term hystero-epilepsy, although epilepsy has no part in its make-up, unless both diseases occur in the same patient, as may be the case. When they do, however, the attacks are separate and distinct in time and characteristics. The typical attack is preceded several days by prodromes, consisting of changes in mood, lack of concentration, depression, and irritability As the paroxysm approaches a more distinct emotional element appears: tears and laughter are easily excited, and alternate without apparent cause. Hallucinations may also be present. Sometimes the patient becomes maniacal. Some of the various stigmata, if they have not been present, appear. The fit may be ushered in by the *globus hystericus*, which consists of the sense of a ball rising in the throat, attended with a sense of suffocation; and the clavus, the peculiar headache of hysteria (p. 102), which may be accompanied by ringing noises in the ears and ovarian hyperesthesiæ.

The **typical convulsion**, as described by Charcot and his followers, is divided into four periods—*i. e.*, (1) the epileptoid; (2) the period of grand movements; (3) the period of passionate attitudes; (4) the period of delirium.

The *first stage* may come on gradually or suddenly. The face becomes pale, the eyes staring, and the patient falls or rather sinks gradually to the floor. It is rare for injury to be caused by the fall, as in epilepsy. Sighs, groans, or exclamations may be uttered, and the body becomes rigid, with a tendency to opisthotonos. The face is slightly congested, and there may be some frothing at the mouth. In

this form of attack consciousness may be completely lost. Following this stage severe clonic movements, more pronounced than those of epilepsy and embarrassed respiration, occur. This is followed after a few minutes by a brief period of more or less complete relaxation, when the *second stage* is ushered in.

In this most intense rigidity of all the muscles is seen, and marked opisthotonos occurs. After a few moments this is followed by rapid flexions of the upper part of the body, throwing of the whole body from side to side, or the legs are carried to the vertical position and then violently dropped again, or more irregular, incoördinate, and non-purposive movements may occur. In this stage there is no facial congestion, and the respiration is about normal.

In from five to fifteen minutes the *third stage* begins, in which the emotions not under control are expressed by gestures. Every emotion that chances to pass through the brain is represented by its appropriate gesture. During this period hallucinations are probably present. The subjects point to objects or appear to be listening to sounds. They imitate various religious ceremonies, and the representation of the different emotional states—viz., fear, anger, remorse, sensuousness, etc.—is vivid and varied. During this stage, also, the patient may sing or give utterance to expressions in harmony with the mental state. Sensory stigmata are present.

In the *fourth stage* the patient is more or less conscious of his surroundings. Illusions and hallucinations, usually associated with vision, are common. The patient talks incessantly about various experiences of past life. Sermons may be preached or poems recited. The cries of animals may be mimicked. This stage may continue for an indefinite period, sometimes for days, sometimes merging into either mutism, trance, or lethargy. In addition to sensory stigmata, motor disturbances, as paralysis or contractures, may develop.

The attack **as seen in this country** is usually atypical and abortive. It may consist of violent alternate weeping and laughing, or, in addition to this, there may be a mild degree of rigidity with slight clonic movements. In this form of

attack consciousness is not lost, but perverted. The patients often describe it by saying that they can hear what is being said but are powerless to move or answer. Partly developed third and fourth stages—catalepsy, lethargy, or somnambulism—may follow, and also any of the various motor or sensory stigmata.

The typical convulsion exclusive of the fourth stage lasts from one-quarter to half an hour, and is known as *hysteria major;* the abortive attack is known as *hysteria minor.*

The *third stage,* that of passionate attitudes, may occur independently of the other stages, and is known as ecstasy.

Hysterical somnambulism also at times occurs independently. It must not be confounded with somnambulism proper, which is an affection of sleep. It is also a modification of the third stage; the patient remembers what occurred during the attack, which is not so in true somnambulism. Patients may also wander away from home, remaining absent for several days, during which period they perform purposive and logical acts of which they have no recollection afterward. Epileptics may have similar attacks (p. 438).[1] *Catalepsy* is a condition of both mental and motor inertia. In it the limbs retain for long periods the position in which they are placed. When bent they yield like a lead pipe. The patient is apparently insensible to external impression, and the somatic functions are sluggish. It also is a variation of the third period. It occurs also in some forms of insanity (p. 553), and can be induced by hypnotism.

Lethargy may also be a part of the major attack or it may occur independently. In it the patient lies in a semi-stupor, with closed eyes, the eylids presenting fine fibrillary tremors. This may last for hours.

Trance is an intensification of lethargy. In it the brain functions are exercised to the most limited extent compatible with the maintenance of life. This condition may endure for many weeks. During the attack weight is lost. Temperature, pulse, and respiration are not much affected.

[1] Patrick, Jour. Nerv. and Ment. Dis., 1907, p. 353.

Pressure over the hysterogenic zones may either cause or abort an attack of hysteria major.

Interparoxysmal Symptoms.—These have been called the *stigmata*. They are divided into sensory, motor, visceral, and psychical:

Sensory.—The alterations of sensation that may occur are its loss or diminution, increase (hyperesthesia) and perversion (paresthesia.)

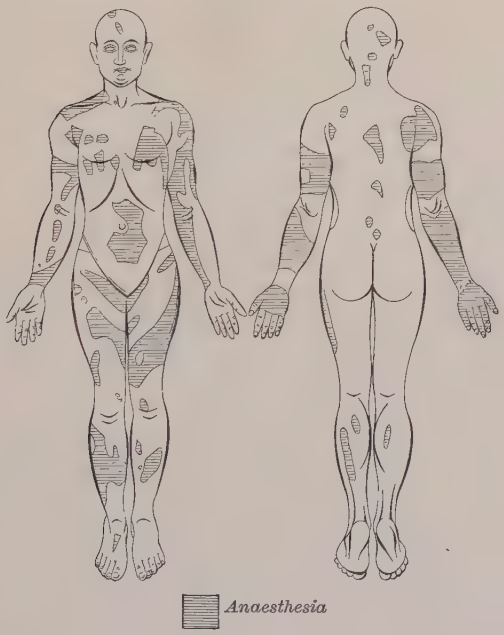
Some form of **anesthesia** is the most common of all the stigmata. It occurs in several forms—viz., *hemianesthesia*, which extends from the crown of the head to the soles of the feet, often involving the mucous membranes, subcutaneous tissues (even the nerves), and special senses. The area stops sharply at the middle line. Vasomotor changes often accompany it, and a pin-prick will not bleed. The limbs may also become edematous and mottled (blue edema). The sharp line of demarcation, the contraction of the visual field, the existence of the lacrymal reflex (secretion of tears when the conjunctiva is irritated), the involvement of the special senses, distinguish hysterical from organic hemianesthesia (p. 48). In the latter form also hemianopsia may be present, and a more or less pronounced motor palsy, showing the characteristics of a central organic lesion (Babinski phenomenon, characteristic gait, etc.), is apt to coexist. Hysterical anesthesia also may vary in its intensity and location from time to time, and may often be made to disappear by suggestion. In testing these patients they should not be asked if they feel a stick or a touch as the case may be, but should be asked what they feel. The liability to cause symptoms by suggestion must always be borne in mind.

Another form is that in which the anesthesia occurs in *scattered patches* assuming various shapes (Fig. 118). They frequently change their position from day to day. A common variety is the segmental form, in which the anesthesia is limited to one limb, or part of a limb. It presents a sharp line of demarcation, often resembling a stocking or a glove (Fig. 119).

Rarely the *entire person* may be anesthetic. *All forms* of

sensation—*i. e.*, tactile, pain, temperature, and muscle sense —may be deficient, or only one of them. According to Pitres, the anesthesia of hysteria is never isolated tactile anesthesia, one or more of the other forms being always combined with it. That most commonly observed is loss of all forms, followed, according to frequency, by analgesia, thermo-anesthesia, combined tactile and thermo-anesthesia,

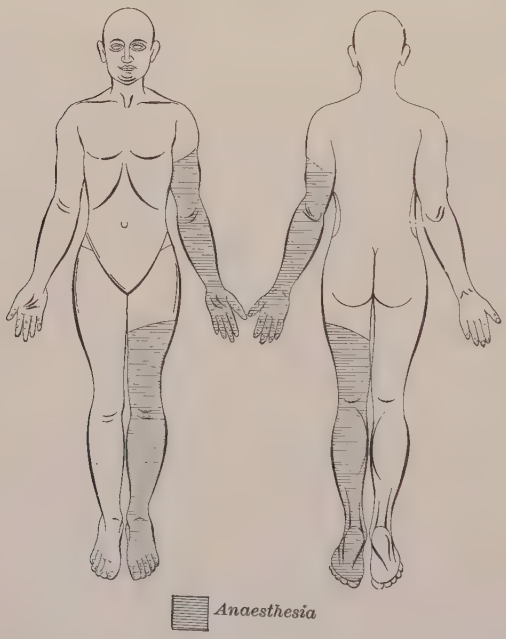
FIG. 118



electro-anesthesia, and loss of all modes except electro-anesthesia. The *degree* of anesthesia may vary from slight diminution in acuteness of perception to absolute loss. It is marked after a paroxysm. Patients are often unaware of its existence, and it should always be searched for. While common, it is not present in all cases. In Briquet's series sensory loss in some degree was found in 60 per cent.

Allochiria (p. 48) is a symptom of hysteria, as are also *achiria* and *synchiria* (p. 49). A group of peculiar perversions of sensation sometimes found have been termed *phrictopathic sensations.* They consist of *abnormal persistence,* the sensation persisting from six to sixty seconds after the stimulus is removed; delay in perceiving the sensation, as much as several seconds elapsing between the stimulus and

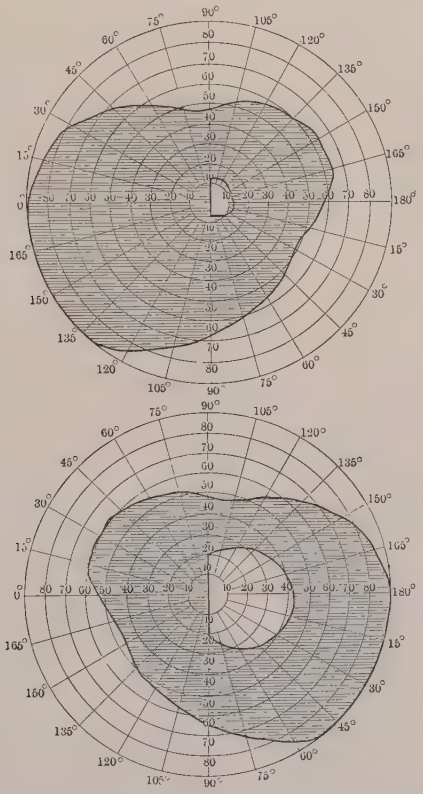
FIG. 119



its perception (this symptom is also found in organic disease, notably tabes); a sudden motor response or start resembling a true reflex; impairment of the sense of personal ownership, *i. e.,* the part touched feels as if it did not belong to the patient; a disagreeable quality of the sensation, whatever the stimulus the sensation, while not painful, is extremely disagreeable; in other words, is *paresthetic*

(p. 47). The skin reflexes in the anesthetic cases are sometimes but not always absent or lessened.[1] The special senses are frequently involved, that of *sight* being most common. It may be totally abolished or partially obscured.

FIG. 120



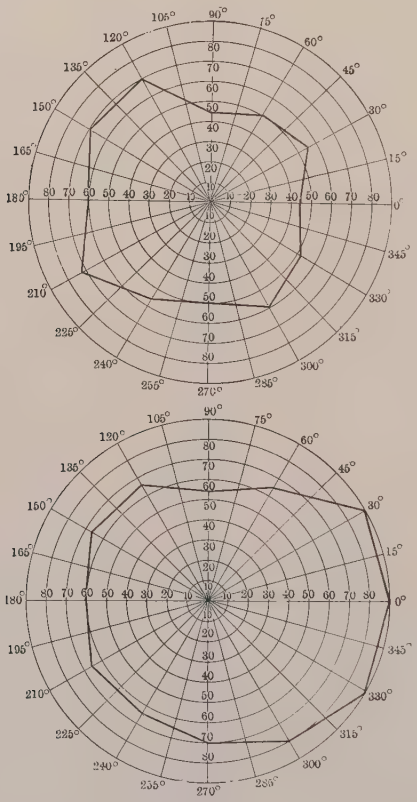
Diagrams of fields of vision in a case of hysterical homonymous hemianopsia (Lloyd.)

Complete blindness is rare; it may appear suddenly and disappear equally so. On the other hand, it may persist a

[1] Knapp, Jour. Nerv. and Ment. Dis., February, 1910, p. 93.

long time. It is sometimes unilateral. When subjected to proper tests, it can usually be proved that patients with unilateral blindness really see with both eyes; for instance,

FIG. 121

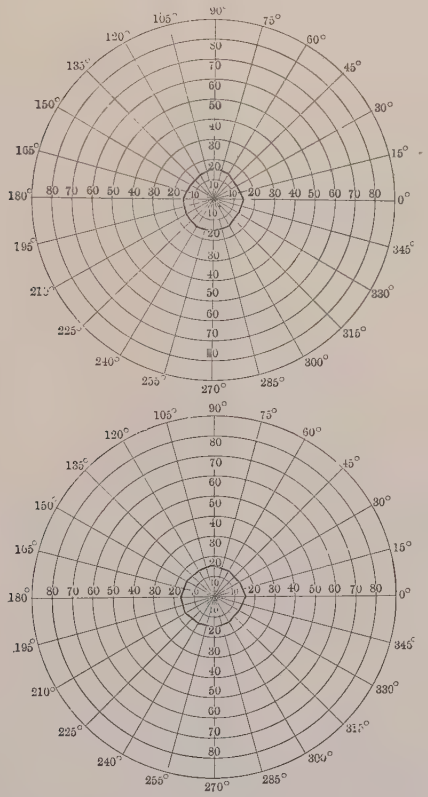


Irregular contraction of the visual field in one eye in traumatic hysteria. (Knapp.)

if a stereoscope is looked through, the picture appears just as it should when seen by means of both eyes, and not as if only seen with one. This is explained by the fact that the loss of sight is purely psychical—*i. e.*, the sensory images

while perceived do not enter into that chain of associated ideas which constitutes the individual's personality; and it is not necessarily evidence of malingering.[1] According to

FIG. 122



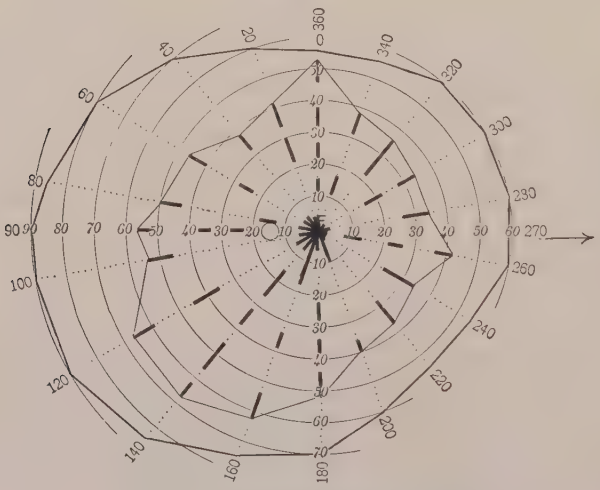
Marked contraction of both visual fields in hysteria. (Gilles de la Tourette.)

Harlan, some of these cases are transiently absolutely blind. Partial blindness is more common, and is indicated by abnormalities of the visual field. Rarely there may be found a

[1] See article by Prince, Amer. Jour. Med. Sci., vol. xciii, p. 157.

central area in which nothing is seen, while in the periphery sight is retained (central scotoma). Equally rare is homonymous hemianopsia, distinguished from that due to organic disease by the presence of hysterical hemianesthesia and involvement of other special senses on that side, and the mucous membranes, and great contraction of the preserved fields (Fig. 120). One of the most common of the sensory stigmata is concentric narrowing of the field, which may be round, oval, or irregular in outline (Figs. 121 and 122).

FIG. 123

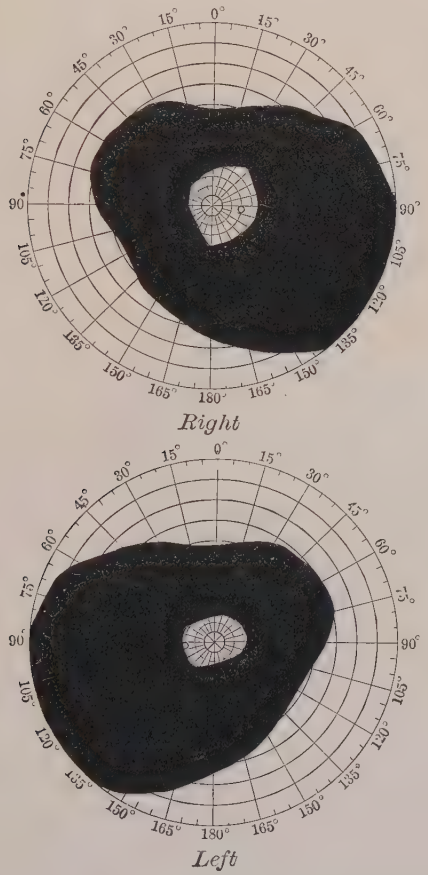


Left field. Oscillating field. (Wilbrand.)

The shifting and exhaustion type, in which it is constantly changing during the examination, seen in neurasthenia (p. 448) may also occur. Another type is the "oscillating," in which the test object appears and reappears several times in the same meridian (Fig. 123). The fatigue spiral field (p. 448) may also be found. Another form is the "tubular field," in which the contracted field remains the same size no matter at what distance the point of fixation is placed (Fig. 124). The perception of colors may be affected;

either lost (achromatopsia); or what is more common, perverted (dyschromatopsia) Fig. 126, or rarely there may

FIG. 124



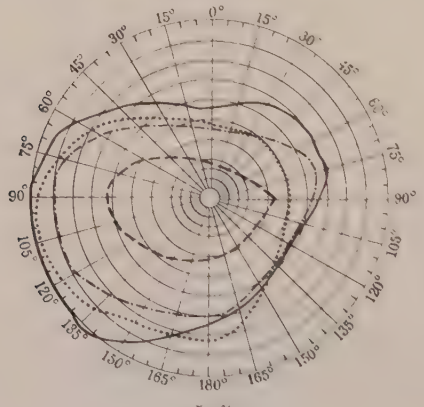
Hysterical girl. Patient of Dr. J. William White. Typical tubular contracted field; no change in size at 30 cm., 1, 2, 3, and 4 meters. (Posey and Spiller.)

be an enlargement of the color fields (hyperchromatopsia) (Fig. 125). In the normal eye colors are not perceived with

equal acuteness, the normal order being blue, yellow, red, green, and violet. In hysteria this order may be changed, and red be perceived before blue, and so on (Fig. 126). Instead of this, the color fields may be concentrically narrowed in the order of their normal position. Hysterical deafness frequently accompanies hemianesthesia. When present it responds to the tests for deafness of nerve origin (p. 58). It is rare for it to be either isolated or complete.

Smell and *taste* may also be impaired; usually unilaterally, as an accompaniment of hemianesthesia.

Fig. 125



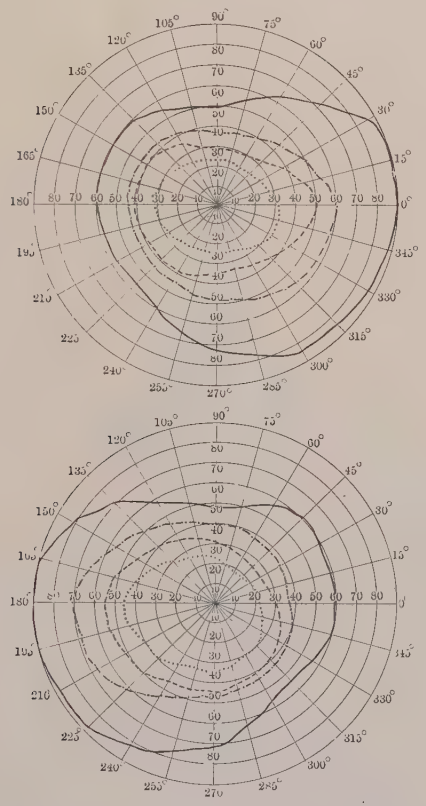
—.—.— = red, ... = green, – – – – = blue.

Hysterical hyperesthesia. Patient of Dr. S. Weir Mitchell. Enlargement of color fields and partial reversal of red and blue lines.

Hyperesthesia is an important symptom, as it is the cause of the so-called *hysterogenic zones*. These are localized hypersensitive areas, pressure upon which will excite the paroxysmal manifestations, especially convulsions, and will also cause them to cease when developed. They are most commonly found over the ovaries, on the breasts, along the spine, and on the trunk beneath the ribs. These areas may

also be the seat of spontaneous pain, and hence simulate local inflammations. This is especially true of joints, organic

FIG. 126



—.—.— = red. – – – – = blue. ..... = green.

Diagrams of fields of vision in a case of hysteria, showing normal form fields and reversal of the red and blue lines, the red field being largest in extent. (Mitchell and de Schweinitz.)

inflammation of which may be closely simulated. Hysterical joints are not associated with deformity, shortening, development of pus, or elevation of temperature. There are usually

marked contractures, more pronounced than the degree of muscular rigidity common about inflamed joints; the pain is more diffuse, and the mental and physical stigmata of hysteria, as paralysis, anesthesia, and a tendency to exaggeration in the account of the symptoms is apt to coexist. Under ether the joint will be found to be freely movable. Of course, as in other conditions, hysteria may be added to an organic affection. Various forms of neuralgic pain occur, as the clavus, already described; in the female breast, which may be accompanied by great tenderness and some swelling; and in the occiput, back of the neck, and along the spine. Retinal hyperesthesia may also occur associated with dread of light, profuse lacrymation, blepharospasm, and neuralgic pains in the supra-orbital and neighboring regions. This condition has been termed "hysterical asthenopia."

Paresthesia, when present, usually takes the form of flashes of heat or cold; sensations of insects crawling over the body, which may be also associated with hallucinations; of numbness, not dependent upon anesthesia; of prickling, tingling, burning, and the like (see also p. 462).

Motor.—These consist of *paralysis*, *contracture*, *tremor*, and *incoördination*.

Paralysis may take the form of either monoplegia, hemiplegia, paraplegia, diplegia, or be limited to a few muscles or muscle groups. It may be either caused or aggravated by a previous convulsion, or follow, independently of this, any of the causes mentioned under etiology. *Contracture* is frequently, but not always, associated with it. The degree of paralysis varies from slight weakness (amyosthenia) to complete loss of power. The deep reflexes are usually somewhat increased[1] or normal and the skin reflexes may be diminished if sensory loss coexist. The loss of power frequently develops either gradually or suddenly, and may disappear suddenly, or it may persist for a long time.

Monoplegia is generally accompanied by anesthesia, which assumes the glove-like or segmental form, and does not

[1] Whether true ankle-clonus is ever due to hysteria or not is a disputed point. A pseudo-clonus in which the movements are not so persistent as in true clonus does occur. The knee-jerks may be lost in hysteria.

follow the distribution of the nerves, as does anesthesia due to peripheral nerve lesion.

Beevor states that in functional paralysis the antagonistic muscles do not relax when the active muscles contract; in fact, they may contract first, which causes the to-and-fro

Fig. 127

Hysterical paralysis of the leg. (Icon. de la Salpêtrière.)

hesitating movement often seen when attempts at movement are made in such cases (p. 43). The test of Hoover[1] may also be employed; this depends upon the fact that if a normal

[1] Jour. Amer. Med. Assoc., August 29, 1908, p. 746.

person laying on the back elevates one leg the heel of the other is pressed downward with equal force, this can be detected by placing the hand under the heel. If organic paralysis exists and an attempt is made to elevate the paralyzed leg, the heel of the sound limb is similarly pressed downward. In either simulated or hysterical paralysis it is not. Also if an attempt to raise the sound leg against opposition is made the pressure downward of the paralyzed leg is proportional to the amount of weakness.

In hysterical *hemiplegia* the weakness is generally more marked in the leg than in the arm. The face and tongue are very rarely, if ever, paralyzed, but these muscles may be affected by contracture. In walking the leg is dragged after the patient, and is not swung in the arc of a circle, as in organic hemiplegia (Fig. 127). There is often anesthesia, either involving the same side (hemianesthesia) or in disseminated areas. An important diagnostic sign is the fact that the toe is flexed in the normal manner when the plantar reflex is tested, and not as it is in organic hemiplegia. (See Babinski Reflex.)

Paraplegia may resemble very much that due to organic causes, but it differs in the mode of onset, the sphincters not being involved; absence of trophic symptoms; normal or but slightly increased reflexes, absence of the Babinski reflex, and in the fact that the area of anesthesia, if present, does not correspond to that which would be caused by an organic lesion if the motor paralysis present were due to such.

Local paralyses may affect a muscle or a group of muscles of a limb, or those of the eye, larynx, pharynx, or esophagus.

Paralysis of the ocular muscles, intrinsic or extrinsic, is rare, but does occur. A curious symptom is monocular diplopia or polyopia, in which one eye sees two images. It may also be due to a corneal deformity, and then, of course, is not hysterical.

Flaccid ptosis, described on p. 144, may occur in hysteria. Pseudopalsies due to spasmodic conditions are more common, and interferences with accommodation, rigidity of the pupil, complete or partial, blepharospasm, pseudoptosis

(Fig. 128), apparent palsies of the external recti, and even conjugate deviation may be so produced. The diagnosis of such conditions depends upon an expert ophthalmologist.

The vocal cords are often paralyzed, producing aphonia; a slight cold or sore throat may be sufficient to cause it. It is usually bilateral, and the adductors are the muscles affected (p. 147). This may be associated with a whispering

FIG. 128

Hysterical spastic ptosis. (Wilbrand and Saenger.)

voice or it may be completely lost. Rarely the abductors may be involved. Anesthesia of the laryngeal mucous membrane may coexist, and, if present, is a valuable symptom. In paralyses of the pharynx and esophagus there is interference with deglutition and a sense of constriction. Examination with a bougie will fail to show evidence of stricture.

Often in hysteria the stomach and intestines become greatly distended with gas, forming apparent tumor (phan-

tom tumor) or simulating pregnancy. Probably, in some instances at least, this is due to paralysis of the muscles of the intestine.

Contracture is a characteristic symptom of hysteria, and usually, but not always, is associated with paralysis. It may involve all the muscles of a limb or only a few of them. Its onset may be sudden or gradual; most frequently the former.

Fig. 129

Hysterical contracture. (Methodist Episcopal Hospital, Philadelphia)

It often disappears suddenly and reappears in another part. It is very obstinate and difficult to overcome. It may not relax during sleep, but does so under an anesthetic. It may be painful; the deformity often assumes forms not common to those due to organic disease; compare that seen in Fig. 129 with the contracture due to organic hemiplegia in Fig. 60.

Contractures also occur in the muscles of the face, tongue, eyes (p. 472), and neck. They usually produce deformities not similar to those which would be produced by a hemiparesis of these muscles accompanying organic hemiplegia; thus the tongue may be protruded away from the paralyzed side, instead of toward it, as in organic hemiplegia. Torticollis would be produced by contracture of the neck muscles. Cases have been reported in which, associated with other stigmata of hysteria, there have been clicking noises, which are audible to a person listening a few inches from the ear. These have been thought to be due to clonic spasm of the soft palate and pharyngeal muscles.

Tremor most frequently occurs in cases due to traumatism. It may be rapid or slow, and often occurs as an intention tremor, simulating that of multiple sclerosis. Cases have been observed in which the arm swings like a pendulum.

Sharp, quick, muscular contractions, producing movements resembling choreic movements, are sometimes seen. They differ from choreic movements in that they are much quicker and more coördinate, while other stigmata of hysteria will be present.

Incoördination may vary from a slight loss of control over either all or a few muscles to the symptom complex known as *astasia-abasia*. This is most frequently seen in children, and is characterized by inability either to stand or walk, while the muscular strength of the legs and ability to execute coördinate movements is preserved when the patient reclines or sits. Anesthesia may or may not be present. *Akinesia algera* is a condition in which all voluntary movements are associated with intense pain, without any inflammatory condition to cause it.

Visceral Symptoms.— Among visceral symptoms due to hysteria are vomiting; cardiac, vascular, and vasomotor affections; rapid respiration; pyrexia; cough; yawning; phantom tumors and anuria.

Hysterical vomiting is characterized by the absence of nausea, the food being regurgitated without effort. In some cases it does not reach the stomach at all. Sometimes there is merely prolonged refusal of food and absence of

vomiting. Such patients may become extremely emaciated. The usual sudden onset after exposure to a case of hysteria, character of the vomiting, and the presence of other hysterical stigmata distinguish it from that due to organic disease.

Symptoms pertaining to the circulatory apparatus consist of either rapid or slow pulse; flushings; edema, which does not pit on pressure, the skin over it being normal or blue in color; erythema; or the smaller vessels may be contracted, as evidenced by the absence of bleeding when the anesthetic limb is punctured.

Rapid respiration is not necessarily accompanied by rapidity of the heart, and there is neither dyspnea nor cyanosis. It may be as high as 90 to the minute.

Hysterical fever may occur without other symptoms. When accompanied by pain it simulates other affections, as meningitis, or other organic disease. Only the prolonged course of the disease, some atypical symptom, or development of typical hysterical stigmata enable the diagnosis to be made.

Hysterical cough is persistent, dry, and brazen, and does not appear to annoy the patient. Paroxysmal gaping is a rare symptom.

Anuria is not a common symptom. An excessive flow of urine (polyuria) is more common.

Psychical Symptoms.—These are the dreamy and trance-like states mentioned on p. 459. They may occur in connection with convulsive attacks or independently. The Ganser syndrome may occur in hysteria. When present the patients act like children, give short and snappy answers, laugh without cause, imitate the cries of animals, and in answering questions give indirect or half answers, and frequently contradict themselves. It is also found in epileptic dream states, alcoholism, and dementia præcox.

Diagnosis.—While hysteria may simulate all diseases, it also possesses certain peculiarities which should enable us to recognize its existence. There will always be found something not typical of the organic disease but which is peculiar to hysteria.

These peculiarities have been detailed under the descrip-

tion of various symptoms. The points of distinction between the epileptic and hysterical convulsion have been given on page 441; between neurasthenia and hysteria on page 451.

A striking peculiarity of hysteria not found in any other disease, excepting syphilis of the nervous system, is a *sudden appearance*, *disappearance*, and *shifting* of the symptoms. This, of course, does not occur in all cases; but when it does, it is suggestive. The distinction between syphilis and hysteria should be easily made by paying attention to the existence of the stigmata above mentioned. It must not be forgotten that hysteria may coexist with many organic diseases of the nervous system and forms of insanity.

Prognosis.—This is doubtful; death is rarely caused by hysteria; but the symptoms may obstinately resist all treatment. On the other hand, some mental impression or other agency may cause a sudden recovery.

Treatment.—In the treatment of the disease the tendency to receive suggestions must be remembered. To utilize suggestion, hypnotism is not essential; all that is necessary is first to gain the patients' confidence, and then continually to impress upon them that certain effects will result from certain causes. Electricity, especially the static form, is a useful adjuvant in impressing the patient. Massage is also of service. Some unpleasant form of treatment may be of service; for instance, in a robust patient cold douches or plunge baths, or hot and cold douches followed by frictions, are often useful. The actual cautery may also exert a good influence. The general condition of the patient should be built up and supported, and drugs should be used only to exert that end. The prolonged use of sedatives is harmful.

In all cases it is of advantage—often, in fact, a necessity—to remove the patient from the influence of sympathizing friends and relatives. Hence it is that the rest treatment of Weir Mitchell is often productive of satisfactory results. The training camp (p. 453) is a valuable plan of treatment. In suitable cases (p. 456) the analytical or "cathartic" method of Freud may be of service; it consists in requiring the patient to endeavor to call into consciousness all

unpleasant experiences, sexual or otherwise, from the earliest years and to tell them to the physician. The dreams of the patient should also be detailed. By an analysis of the thoughts of the patient so obtained the cause of the symptoms is ascertained. The bringing of these buried thoughts to the surface and "getting them out of the system," so to speak, causes a disappearance of the symptom.[1] An hysterical paroxysm can often be cut short by a hypodermic injection of apomorphine or the vigorous use of cold water.

TRAUMATIC NEUROSES

Definition.—Various organic affections of the nervous system may be due to trauma. Thus, definite changes in its structure may be caused by either contusion or crushing of the tissues or by hemorrhages, single or multiple. Injuries, without discoverable external lesion, may cause diminished resistance to the action of infections, and thus give rise to the development of disease from such cause. Epilepsy, multiple sclerosis, myelitis, syringomyelia, tumor of the brain, and, possibly, tabes, although this is denied by many, may be the results of a previous trauma.[2] The *functional disorders*, either *neurasthenia* or *hysteria*, may also be caused by trauma, and when symptoms of either of these or of both combined follow this cause, the patient is said to have a traumatic neurosis. The names "railway brain" and "railway spine" have also been applied.

Etiology.—Traumatic neuroses may be caused by any sort of injury, and seem especially liable to result from an injury which gives rise to jarring or concussion of the whole body. An important element in the etiology is the coexistence of fright or mental shock, and a very trifling physical injury may be sufficient to produce the symptoms if so accompanied. Indeed, mental shock alone may be sufficient. In some

[1] Jour. Abnormal Psychology, June and July, 1909, p. 72; Rev. Neurol. and Psychiat., March, 1910, p. 135.

[2] Schultze, Deutsch. Zeitsch. f. Nervenheilkund, 1910, Bd. xxxviii, S. 238; Abstract in Rev. Neurol. and Psychiat., April, 1910, p. 240.

cases, when blows upon the back are received, actual damage may be done to the intervertebral joints, causing certain symptoms, to be described later. As has been shown under the description of these affections, patients with neurasthenia or hysteria are introspective; they are continually watching their old symptoms and hunting for new ones. The latter also are prone to receive suggestions. Now, as the traumatic cases are often the cause of litigation, they are subjected to examinations by lawyers and physicians and to the worry and excitement which attend all litigation, these factors tend at least to aggravate unconsciously the condition. It must not be supposed that all such cases are subjects of litigation. They frequently occur when such is not thought of or possible.

Symptoms.—If the injury is severe the ordinary symptoms of concussion of the brain (if cerebral), or of ordinary surgical shock if elsewhere, occur at once. If the injury is slight, the patient may be thrown at once into a dazed condition, in which he performs various actions correctly, but may have no remembrance of them afterward; or he may be dazed for a moment, then recover, and, after a greater or less period of time, the symptoms develop. In a few hours, in some cases, the patient may be seized with a spell of hysterical weeping or he may be unable to sleep. In others the symptoms develop gradually, and it may be several weeks before anything noticeably wrong is discovered. The symptoms, when fully developed, consist of various combinations of those described under hysteria and neurasthenia. A rare condition has been described, termed by Dana the "grave traumatic neurosis," and by Knapp "traumatic sclerosis," in which the symptoms are very severe and peculiar mental symptoms occur. These consist of marked depression, sometimes nearly amounting to stupor, loss of memory, emotionalism, lack of attention—in other words, an "inhibition of psychic processes." The face is expressionless. The physical symptoms consist of very marked motor and sensory paralysis of the hysterical type, tremor, etc.[1]

[1] Bailey, Diseases of Nervous System due to Accident, second edition, p. 511.

When there has been either a direct blow upon, or twisting or wrenching of the back, the intervertebral joints or the muscles may be damaged, and the condition known as traumatic lumbago produced. This is characterized by pain in the back, much increased by movement. Pain may also extend from the back around into one or both sides or

FIG. 130

Attitude of a patient with traumatic lumbago. (Dercum.)

into the legs. The muscles are in a state of rigidity, which is increased by attempts at movement. The patient walks slowly and deliberately, holding the back stiffly (Fig. 130). Pain is elicited by deep, hard pressure over the affected areas, and Mannkopf's sign, which consists of an increase in the pulse rate while pressure is being made upon the tender area, may sometimes be of service as an objective sign of

pain. Pain may, however, be present and this sign absent. The symptoms are not always so pronounced as have just been described.

Diagnosis.—It is necessary first to determine that the patient is not simulating. To do so, repeated and careful examinations must be made, taking care not to ask leading questions. The presence of objective symptoms, as contracted visual fields, tachycardia, contractures, etc., would be of great assistance in determining that the condition was genuine (p. 460). When the symptoms are entirely subjective, the decision may be more difficult. It is not often, however, that a layman would be able to detail a group of symptoms characteristic of the genuine condition, and repeat the same without essential variation at different examinations. As a matter of experience, simulation of these conditions is not very common.

Having determined that there is a genuine disorder, it may be necessary to determine that no organic disease of the nervous system is present, or, if a joint is affected, whether the affection is organic or hysterical. For the distinguishing features the reader is referred to the description of hysteria, neurasthenia, and organic diseases of the nervous system.

Prognosis.—The prognosis of traumatic neurosis as to complete recovery is doubtful. Not much improvement can be hoped for while legal proceedings are in progress. After they cease, some improvement, as would naturally be expected, often occurs, but complete recovery cannot safely be promised, although it may, and does often occur (pp. 452 and 477). The grave forms described on page 479 rarely if ever completely recover. Traumatic lumbago is an obstinate affection, and spondylose rhizomélique (p. 503) may develop. Most cases, however, improve in time, although the back may be always a weak spot, liable to develop pain in damp weather or on slight exertion. If put under the proper conditions early, cases of traumatic neurasthenia and hysteria often do well.

Treatment.—This should be instituted early, and the vexatious excitement and examinations incident to legal proceedings avoided if possible. Hence an early settlement

should be advised in preference to a jury trial, even if the compensation is likely to be less. The patient should be encouraged and conversation referable to the accident or symptoms suffered from avoided. The rest treatment of Weir Mitchell (p. 93), when it can be obtained, is the preferable form of treatment. If it cannot be, other measures advised for the treatment of neurasthenia and hysteria may be employed.

For traumatic lumbago, rest in bed, gentle massage of the affected muscles, the galvanic current, the anode being placed over the tender points, should be employed. In extreme cases, counterirritation, preferably with the actual cautery, may be tried (p. 504).

OCCUPATION NEUROSES

Definition.—An occupation neurosis is a condition occurring in persons whose business requires the constant repetition of certain movements, which, in most cases, are fine and complicated, but may be simple and coarse, in which they lose the power of performing these movements, while all other movements can be performed as usual.

Etiology.—It is most apt to occur in neurotic individuals, especially those who are neurasthenic, and anything which acts as a cause in producing that condition may act as a predisposing cause in the development of an occupation neurosis. It is more common in men than in women.

The most common cause of this condition is excessive writing, and when so produced is known as *writers' cramp*, or writers' or scriveners' palsy.

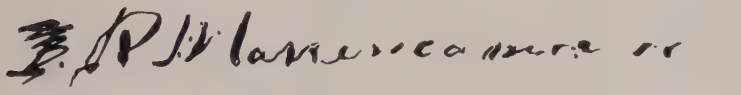
Symptoms.—*Writers' cramp.*—Three types have been described—*i. e.*, the *paralytic*, the *spasmodic*, and the *tremulous*. They are rarely sharply defined, the symptoms of each more or less commingling. Usually the first symptom noticed is, that after writing a while a dull ache in the wrist and metacarpal joints, and a sense of fatigue is felt. After ceasing to write, this passes away to return again at the next attempt. Finally, these symptoms remain permanent and

may extend up the arm even to the scapula. Soon motor symptoms appear; the patient cannot control the pen; it digs into the paper or flies about in various directions; or it is grasped tightly and there is inability to move it in any direction (Fig. 131); or the pen is dug tightly into the paper, making characters such as are seen in Fig. 132. More rarely the pen drops from the fingers. The spasm is tonic not clonic. Sometimes the muscles of the arm and shoulder may be affected in addition to those of the thumb and fingers. If tremor occurs, the handwriting is shaky and tremulous; it is usually only present during the attempt to write, but in advanced cases may be continuous. There is usually some degree of spasm also present.

FIG. 131

Handwriting in case of writers' cramp. The patient could not make another letter in this attempt to write his name.

FIG. 132



Handwriting in writers' cramp.

As a rule, all movements excepting those employed in writing can be well performed. Sometimes, however, there may be involvement of some others, as in a patient reported by Gowers, who also lost the ability to shave. Tender points are often found over the nerve trunks and paresthesia may be present. The general condition of the patient is apt to be neurasthenic.

There are a number of other occupations which may produce similar symptoms, only differing in the character of the movements effected. They are principally found in seamstresses, telegraph operators, piano players, typewriters,

and blacksmiths. Cases have also been reported in drummers, money counters, weavers, compositors, engravers, dancers, cigarette makers, painters, and others.

Diagnosis.—The early stages may be mistaken for neuritis; but the fact that all other movements can be well performed eliminates that.

Prognosis.—The prognosis as regards recovery is not very good, especially if the case is of long standing. Recoveries, however, do occur.

Treatment.—The most essential thing is the prohibiting of the performance of the affected movements. If this is impossible, in some occupations the use of the left hand may be learned, although this may also become affected in time. In writing, a soft pen, thick holder, and a free-arm movement should be employed. Such precautions are also of value in preventing the appearance of the disease. The general health must be built up by appropriate measures. (See Neurasthenia.) Galvanism, the anode, applied labile over the muscles of the arm for five minutes, followed by a constant current passed down the arm for ten minutes, often gives relief. The static breeze or high-frequency effluve may also be of service. Massage is useful. Outdoor exercise and local gymnastics, the fingers made to perform various movements unlike those affected, often gives excellent results. The various appliances which have been invented to enable the patient to write, soon lose their efficiency.

TETANUS (LOCKJAW)

Definition. — An acute or subacute infectious disease, characterized by tonic spasm, with violent exacerbations, without loss of consciousness.

Etiology.—It occurs "idiopathically" or after trauma. In certain localities it occurs in epidemic form among newborn children (trismus neonatorum). It is more common in hot than temperate climates. Most cases follow wounds, usually punctured, of the hands and feet. A very trifling wound may be sufficient. It has followed the administration of diphtheria antitoxin which was derived from infected horses,

many of the cases following vaccination, however, are due to lack of care and infection of the wound. It also occurs in women after childbirth. It frequently follows wounds made by blank cartridges. The essential cause is the *tetanus bacillus*.

Pathology.—No characteristic lesions have been found. The disease is due to infection by a virus produced by a bacillus which is found in the earth, in putrefying fluids, and manure.

Symptoms.—The symptoms of tetanus generally appear within ten days after the injury. A feeling of stiffness in the neck or difficulty in mastication is first complained of. Gradually a tonic spasm of the muscles of mastication develops, producing the condition of *trismus*, or lockjaw. The eyebrows may be raised and the angles of the mouth drawn out, producing the so-called *risus sardonicus*. By degrees the muscles of the body are involved. During the height of the spasm the body assumes the position of opisthotonos; or, less commonly, the body and limbs may be rigid (orthotonos) or flexed to one side (pleurothotonos); or spasm of the abdominal muscles may cause it to be bent forward (emprosthotonos). During the paroxysm the respirations are rapid, and spasm of the glottis may cause asphyxia. The slightest irritation is sufficient to cause a spasm, which lasts for varying periods, but complete relaxation does not occur in the intervals. The temperature may be either normal or considerably elevated (105° to 106°). Consciousness is preserved, and severe pain attends the paroxysms.

A form known as *cephalic tetanus* may result from wounds on one side of the head, and is characterized by stiffness of the muscles of the jaw, paralysis of the facial muscles on the same side as the wound, and difficulty in swallowing.

Diagnosis.—Tetanus resembles strychnine poisoning, but in that the jaw muscles are not involved first, and there is no rigidity between the paroxysms, or elevation of temperature. The points that distinguish it from tetany are given under the account of that disease.

Prognosis.—The prognosis is bad. Chronic or subacute cases—those in which the symptoms develop late, in which

the spasms are localized to the muscles of the neck and jaw, and in which fever is absent—frequently recover. It is worse in traumatic cases than in the "idiopathic." Cases that live over four days usually recover.

Treatment.—The wound, if found, should be excised and antiseptic treatment employed. The patient must be kept in a darkened room and all sources of irritation avoided. If food cannot be taken by the mouth, he may be fed by nutritive enemata. The paroxysm should be controlled with chloroform. Either morphine, chloral hydrate, or the bromides should be given in full doses. Tetanus antitoxin has been used with some success. It may be given by injection either subcutaneous, into the spinal canal or the main peripheral nerve near the situation of the primary wound. 5 c.c. of a 25 per cent. solution of magnesium sulphate injected into the subarachnoid space has been of service. Prophylaxis is of most importance. For this and further details concerning treatment the reader is referred to works on surgery.

HYDROPHOBIA (RABIES)

Definition.—An acute infectious disease of carnivorous animals, which may by inoculation be transmitted to man and other animals.

Etiology.—It is more common in some countries than in others; in this country it is rare. The dog is particularly susceptible. The nature of the poison is unknown. It is found in the nervous system and some of the secretions, notably the saliva. All persons bitten by rabid animals do not develop the disease. Bites through clothing are less dangerous than those of exposed parts, as the hand or face.

Morbid Anatomy.—The vessels of the brain and cord are congested with perivascular exudation of leukocytes and minute hemorrhages. These are most intense in the medulla. Babes has shown that in rabies a collection of embryonic cells is found about the central canal of the cord and medulla, especially about the motor cells. These collections of cells have been termed "rabic tubercle." Van Gehuchten and

Nelis have also found in the peripheral cerebral and sympathetic ganglia, and especially in the intervertebral and plexiform ganglia of the pneumogastric nerve a destruction of the nerve cells, brought about by new-formed cells derived from the capsule, which appear between the cell body and its endothelial capsule, and which finally completely occupy the entire capsule.[1] Of still more importance is the finding of irregular bodies, probably protozoa, in the cells of the cerebellum, cerebral cortex, pons, and cord. They are termed Negri bodies, after their discoverer. Reichel found them in 99 per cent. of cases known to be positive, and none in negative cases. The pharynx, larynx, trachea, and bronchi are congested, and the mucous membrane of the stomach is hyperemic and often covered with mucus.

Symptoms.—The period of *incubation* varies from six weeks to three months. There is first a premonitory stage, in which there may be irritation, pain, and numbness about the bite. There are headache, loss of appetite, depression of spirits, and elevation of temperature. There are also irritability and sleeplessness. The voice may be husky and some difficulty in swallowing be experienced.

Gradually great excitability, restlessness, and hyperesthesia develop. The slightest peripheral irritation causes muscular spasm. The muscles of the larynx and mouth are particularly affected, and the attempt at swallowing causes painful spasms of these muscles. During the spasms the patient may be maniacal, in which he may attempt injury to other people. This, however, is unusual. In the interval between the spasms the patient is quiet and rational. The temperature is usually elevated.

In from one to three days the patient passes into the so-called paralytic stage. He becomes quiet, the spasms cease, unconsciousness develops, and death occurs in from six to eighteen hours.

Diagnosis.—Animals suspected of having the disease should be kept confined until unmistakable evidences appear. When killed, the medulla should be secured and rabbits

[1] McCarthy and Ravenel, Univ. Med. Mag., January, 1901, p. 766.

inoculated with it. The occurrence or not of the symptoms in the inoculated animal will remove all doubts. Microscopic examination of the nervous system should also be made to determine the existence of the changes described under pathology.

Pseudohydrophobia is comparatively common. It is an hysterical outbreak occurring in a person several months after they have been bitten by a dog. The symptoms resemble somewhat the true disease; but the temperature is not elevated and the symptoms persist longer than do those of true hydrophobia. The patient may bark like a dog, and is very emotional and talks about his condition.

Prognosis.—The prognosis of hydrophobia is fatal. Inoculation by the method of Pasteur appears to have an important influence in preventing the onset of the disease. The wound should early be excised or thoroughly curetted and cauterized. A ligature may be applied above the wound and free bleeding from it encouraged. The treatment of the developed disease is the same as that of tetanus, with the exception that the tetanus antitoxin would be of no service.

TETANY

Definition.—A condition characterized by bilateral tonic spasms, either paroxysmal or continuous, of the extremities, and not attended usually by loss of consciousness.

Etiology.—It is probably due to an infection. Debility, following chronic diarrhea or lactation, and rachitis are the most common causes. It may occur during pregnancy or following infectious fevers. It follows removal of the parathyroid glands and the excessive excretion of calcium salts in the urine present in the disease has etiological importance. It may be associated with dilatation of the stomach, and occur after the introduction of the stomach tube. In certain parts of Europe it occurs endemically.

Symptoms.—The attacks are commonly paroxysmal, lasting a variable time, from a few minutes to a few hours. In the continuous cases the spasm may last days or weeks.

There may be feelings of numbness or pain in the extremities or a general feeling of lassitude for a short time previous to the onset of the paroxysm. In most cases spasms are confined either to the hands alone or to the hands and feet. The fingers are closely pressed together, the thumbs adducted and pressed either firmly against the index fingers (writing posture), or flexed into the palms, beneath the fingers. The hand itself and elbow are also generally flexed. In the lower extremities the toes are strongly flexed and the feet are held in the talipes equinovarus position. The thigh muscles usually escape. In severe cases the muscles of the face may be involved, causing trismus and drawing out of the angles of the mouth. Rarely there may be a slight degree of opisthotonos and fixation of the thorax, with difficult respiration, which is increased if the laryngeal muscles are involved, when the features of laryngismus stridulus may be present. These consist, in the absence of inflammation of the larynx, of arrest of respiration, and congestion of the face due to spasm of the laryngeal muscles, followed by sudden relaxation of the muscles, the air being drawn into the lungs with a high-pitched, crowing sound. It is probably related to tetany. Unless the spasms are severe, there is no pain. In cases due to either extreme debility or dilated stomach there may be loss of consciousness. Usually the mind is clear. The temperature during the attack may be either slightly elevated or subnormal.

In the intervals between the paroxysms, spasms can be induced by a firm, continued pressure on the nerve trunks or vessels (Trousseau's symptom). A slight tap over a nerve is sufficient to produce contraction of the muscles supplied by it; thus, if a slight tap is made over the facial nerve, the muscles supplied by it will be thrown into contraction (Chvostek's symptom). The electrical excitability of the motor nerves is also increased, a weak current causing tetanic contractures; and we may have, instead of the normal formula, AnClC or AnOC > KClC (Erb's symptom).

The sensory nerves are also more irritable to electrical and mechanical stimulation; a weak current or slight pressure over such a nerve as the supra-orbital causing paresthesia

in the parts to which the nerve is distributed (Hoffman's symptom). Attacks may consist of paresthesia and stiffness without spasm.

Diagnosis.—Tetany is distinguished from hysteria by the presence of Trousseau's symptom and the hyperexcitability of the muscles which is peculiar to tetany alone.

From tetanus, it is distinguished by the intermittence and milder degree of the symptoms, and the fact that the spasms begin in the extremities instead of in the jaw muscles.

The disease rarely causes death, excepting when due to gastric dilatation. In these cases the mortality is high. It may last from a few weeks to a few months. It may recur each year for a number of years.

Treatment.—The most essential part of the treatment is that of the cause. In cases following removal of the parathyroid, extract of that gland should be given. Full doses of calcium salts are indicated in all cases, as is also food rich in calcium salts. For the attack, antispasmodics, as bromides, valerian, hyoscine, etc., may be given. In severe cases inhalations of chloroform or morphine may be needed. Gowers advises a dose of digitalis at bedtime for the attacks that occur at night. Ice-bags to the spine and lukewarm baths may also be of service. Lavage with large quantities of saline solution and other measures appropriate for the treatment of gastric dilatation may be beneficial in gastric cases.

FAMILY PERIODIC PARALYSIS

Definition.—A rare disease, of unknown pathology, which attacks members of successive generations of a family, and is characterized by periodical attacks, of a more or less sudden onset, in which there is an extensive, flaccid, motor paralysis, associated with loss of knee-jerk and electrical excitability, and without sensory or psychic disturbance of any sort.

The cause is unknown. In one case studied by Mailhouse[1]

[1] Jour. Nerv. and Ment. Dis., April, 1910, p. 209.

there was a diminished excretion of calcium and magnesium salts and he mentions the possibility of the paralysis being due to their inhibitory influence upon nerve and muscle, when they are retained within the body.

Symptoms.—In the majority of cases the attacks begin between the ages of ten and twenty. In some cases there are prodromata, as feelings of weariness, numbness, or formication, headache, backache, sweating, etc. The loss of power usually begins at night, and gradually progresses for a varying period for several hours to several days before the height of the paralysis is reached. In most cases the legs are first affected; the muscles of the body, arms, and neck in severe cases becoming affected later. Involvement of the cranial nerves is rare. During the attack there is loss of the knee-jerk, and the electrical irritability of the muscles varies from quantitative decrease to absolute loss for both currents. In the intervals between these attacks both knee-jerk and electrical irritability are normal. In some cases, during the attack, cardiac enlargement, with a mitral systolic murmur, which disappears in the normal intervals, has been reported.

The usual duration of the attack is from ten to forty-eight hours, recovery of power taking place gradually. Treatment so far has proved of no avail. The citrates have been recommended.

MYOTONIA CONGENITA (THOMSEN'S DISEASE)

Definition.—A rare disease, occurring usually in members of different generations of a family, characterized by rigidity or spasm of the muscles occurring when their use is attempted after a period of rest. This rigidity passes off in a short time, and does not return while the muscle is being used.

Etiology.—Heredity is the most important factor in the etiology. The affection is usually first noticed in early childhood. The disease is rare in this country.

Symptoms.—The first symptom noticed is stiffness and slowness in the movements of the child, so that he is unable

to take part in games requiring rapid action of the muscles. After a long period of rest, the spasm may be so intense that any movement for a time is impossible. After the spasm passes off, the patient can use the muscles as other individuals, until after the next period of rest. The legs are more often affected than the arms. The mechanical and electrical irritability of the muscle is increased. A quick blow upon a muscle causes a contraction of the fibers, so that a distinct groove may last for ten or fifteen seconds. Firm pressure causes tonic contraction of the entire muscle. The electrical changes, known as the myotonic reaction (p. 79), have been described. The irritability of the nerve is not changed. There is no atrophy; if anything, the muscles are firmer than usual. Myotonia atrophica might be mistaken (p. 283).

No **treatment** seems to be of service.

MYATONIA CONGENITA

(Amyotonia Congenita)

This disease, first described by Oppenheim in 1900, occurs in early childhood and is characterized by a more or less general hypotonia of the muscles. The limbs, especially the lower, are flaccid and motionless, the joints are abnormally movable, and the knee-jerks are diminished or lost. In some cases slight power of movement may be preserved or only weak voluntary contractions may be present, which do not cause movement of the limbs. If the neck and back muscles are affected there is inability to sit up and support the head on the body. The electrical reactions are either quantitatively diminished or absent. The condition is congenital, which distinguishes it from progressive muscular dystrophy (p. 276). It is further distinguished by the fact that it is not a family disease. In anterior poliomyelitis the child has been previously healthy and the onset is acute (p. 257). Amaurotic family idiocy (p. 256) is distinguished by blindness and failure of mental power, which in myatonia

is preserved. Oppenheim believes it due to an arrested development of the muscle. In Spiller's case,[1] the muscles only were found affected. There was, however, a lesion of the thymus gland. Bernhardt[2] has advanced the view that it is due to an arrested development of the peripheral nerves.

The disease is not always fatal, and improvement from the use of strychnine internally and measures to improve the nutrition of the muscles (electricity, massage, etc.) is to be hoped for.

[1] Univ. Penna. Med. Bull., January, 1905.
[2] Neurol. Centralbl., January 2, 1907, p. 2

CHAPTER XV

VASOMOTOR AND TROPHIC DISEASES

RAYNAUD'S DISEASE

Definition.—Raynaud's disease, also known as *symmetrical gangrene* and *local asphyxia*, is due to a vascular change, probably vasomotor, in which there is no organic change in the vessels. It is chiefly seen in the extremities, but may also occur in the internal parts, in which a persistent ischemia or a passive hyperemia leads to disturbance of function or to loss of vitality with necrosis of the parts.

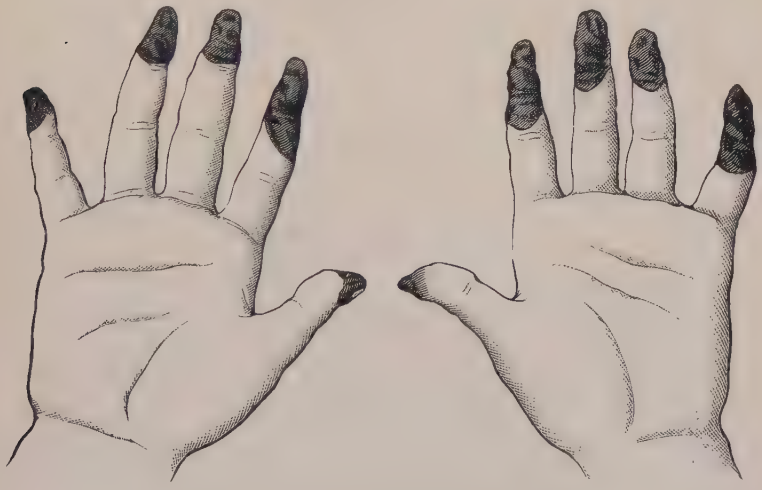
Etiology.—Most cases have occured between the tenth and thirtieth years. It may be a family disease. It is especially apt to occur in neurotic individuals. Shock, fright, exposure to cold and damp, the menstrual period and pregnancy may be exciting causes. Some cases are due to arteriosclerosis, in which the vessels are liable to spasm. These cases must not be confounded with those in which there is an obliterative arteritis (p. 496). It may be associated with migraine and other vasomotor disturbances.

Morbid Anatomy and Pathology.—This is obscure. Neuritis has been found in some cases. The syncope is produced by spasm and consequent contraction of all the vessels, veins, venules, capillaries, and arteries; the asphyxia to the spasm in the venules and veins relaxing first and allowing a backward flow of blood into the empty capillaries; when the spasm in these relaxes hyperemia occurs.

Symptoms.—The fingers and toes are most frequently affected; but the ears, nose, or lips, and patches of skin over various parts of the body may suffer. The parts become pale, bloodless, and cold, resembling, if in the fingers or toes, those of a dead person. The prick of a needle does

not draw blood, and the part feels numb. This stage may last from a few minutes to several hours, and is sometimes attended with constitutional symptoms, as chilliness and general discomfort. The paroxysm may be confined to this stage, or it is succeeded by the stage of local asphyxia, in which the part becomes congested and cyanotic, the blood returning slowly after pressure, and attended with more or less pain. Less commonly the parts after this stage become bright red, hot, and covered with perspiration. If the stage

FIG. 133



Gangrene of fingers in Raynaud's disease. (Dehio.)

of asphyxia continues long enough—several hours—small blebs may appear, followed by ulceration, and finally gangrene may destroy the part (Fig. 133). Hemoglobinuria may develop during an attack, or take the place of one. Mental torpor and transient loss of consciousness have also occurred in some cases. Transient attacks of hemiplegia have been reported. Mania and delusions may also be present. Epileptiform convulsions have occurred.

Diagnosis. *Acroparesthesia* (p. 110) may be mistaken, some indeed have thought that it is a mild form of Raynaud's

disease. Barker and Sladen[1] have described a case in which there was persistent cyanosis with diminution of all forms of sensation in the legs and gangrene of the toes which differed from Raynaud's disease in that the asphyxia was not paroxysmal and not preceded by syncope. The condition especially prevalent among the Russian Jew, in which there is an obliteration of the vessels due to arteritis and thrombosis, and termed by Buerger[2] *thrombo-angiitis obliterans*, closely simulates Raynaud's disease; in this the feet are especially liable to be affected, there is more or less constant pain, often relieved by allowing the feet to hang down, tenderness of the calves, and loss of pulsation in the posterior tibial and dorsalis pedis arteries. *Erythromelalgia* (p. 497) differs in that cold often relieves the symptoms, while heat aggravates them; there is no syncope, and the pain and congestion are aggravated by allowing the feet to hang down or in walking upon them.

Senile gangrene can usually be excluded by the fact that the above conditions usually occur in comparatively young people. *Diabetic gangrene* is excluded by examination of the urine. *Cyanosis* and *gangrene* may occur in diseases of spinal cord, brain, and peripheral nerves, as in hemiplegia, tabes, myelitis, and neuritis. Osler[3] has noticed somewhat similar symptoms caused by cervical ribs.

Treatment.—During the attack of syncope either warm applications or wrapping the parts in cotton-wool should be employed. Nitroglycerin is a most valuable remedy. If the stage of asphyxia is severe the patient should remain in bed with the feet elevated (if they are affected) and the parts wrapped in cotton-wool. If the pain is severe morphine may be given. Massage of the extremities and electricity in the form of the constant current may be of benefit. The latter can be given by placing the affected part in a basin of salt water and placing the cathode in the water while the anode is placed over the spine. The application should last ten minutes or more. Radiant heat is often useful. Cushing

[1] Jour. Nerv. and Ment. Dis., December, 1907, p. 745.
[2] Amer. Jour. Med. Sci., January, 1910, p. 105.
[3] Ibid., April, 1910, p. 469.

has introduced, in cases of syncope, a plan of treatment which has been successful. It consists of applying an elastic bandage to the limb, tight enough to stop the arterial circulation; this is left there several minutes, then loosened, when the affected part will usually become red. The procedure may have to be repeated several times in severe cases. Measures to improve the general health should also be employed. If gangrene occur, amputation becomes necessary. It should be done high up.

Erythromelalgia (Red Neuralgia, Weir Mitchell's Disease).—Definition.—"A chronic disease in which a part or parts of the body, usually one or more of the extremities, suffer with pain, flushing, and local fever, made far worse if the parts hang down." (Weir Mitchell.)

Etiology.—It is more common in men in middle life. Puberty, menstrual disturbance, and the menopause have been mentioned as predisposing causes. It has followed infections, as gonorrhea, rheumatic fever, and syphilis. Exposure to cold and wet, overexertion, and the neurotic diathesis have been given as causes.

Pathology.—Neuritis has been found in some cases; in others the findings have been negative, and they may be due to vasomotor disturbance allied to that causing Raynaud's disease; arteriosclerosis also may be a cause.

Symptoms.—In brief these are redness, pain and swelling aggravated by allowing the affected parts to hang down and relieved by elevating them. The feet are most commonly affected. The redness is more that of an active hyperemia, and a local elevation of temperature may be present. Excessive sweating of the part may occur. Atrophy of muscles has been noted.

Diagnosis.—This must be made from the various affections mentioned under Raynaud's disease (p. 495), all of which it may simulate. It may occur in diseases of the spinal cord and peripheral nerves.

Treatment.—This is usually unsatisfactory. Prolonged rest with elevation of the affected part, massage, cold packs and douches, electricity as advised for Raynaud's disease, except that the anode should be placed in the water,

Cushing's method for Raynaud's disease, p. 496, and radiant heat have all been recommended. If localized to the distribution of a particular nerve it may be excised.

ANGIONEUROTIC EDEMA

Synonyms and Definition.—This disease, also known as acute circumscribed edema, acute non-inflammatory edema, giant urticaria, periodic swelling, Quincke's disease, etc., is characterized by the acute development of circumscribed swellings of the subcutaneous or submucous tissues and commonly attended by gastro-intestinal disturbances. It may be hereditary and is often recurrent. It is a vasomotor neurosis.

Symptoms.—The swellings generally appear without warning and reach their maximum in from one-half to two hours. For a short time before the appearance of the swelling there may be a feeling of malaise and some gastro-intestinal disturbance. The parts usually affected are the face, the lips, tongue, pharynx, genitals, and forehead. The swelling is tense, sharply defined, does not pit on pressure, is not tender, and is whitish or pinkish in color. There is a feeling of tension, and often burning and itching sensations. The swelling lasts from a few hours to several days, and disappears as rapidly as it came, sometimes succeeded by another in a different locality. Scratching or rubbing the skin causes redness and the formation of urticarial wheals. When the tongue, larynx, and pharynx are affected, dyspnea and difficulty in swallowing are caused, and danger to life may result from occlusion of the larynx. Gastro-intestinal symptoms occur in about one-half the cases. They consist of a feeling of uneasiness in the epigastrium, distention, colic, nausea, vomiting, and diarrhea. The secretion of urine may be increased, and contain albumin and hemoglobin. An effusion into the joints may be present. The attacks occur at varying intervals, and the general health in the intervals is usually good. In some of the congenital cases the swelling may be permanent.

Diagnosis and Prognosis.—The disease may be confounded with the blue edema of hysteria and erythema nodosum. The occurrence of some of the stigmata of hysteria, as paralysis of motion or sensation, will distinguish the former. In the latter the swellings are red in color and there is tenderness. The absence of pitting on pressure distinguishes it from edema due to cardiac or renal disease.

Unless the mucous membrane of the larynx and pharynx is attacked, there is no danger to life. Cases not due to hereditary taint frequently recover.

Treatment.—This consists in the removal of the cause, if possible, and the use of vasomotor and general tonics. Strychnine in full doses is the most useful drug. Atropine, given in full doses during the attack and in small doses in the intervals, is also useful. Osler has seen good results from the prolonged use of nitroglycerin. These may be combined with the use of mineral acids, exercise, massage, and tonic baths. Cold and trauma must be especially avoided. If there is an excess of uric acid in the blood, the diet must be regulated, and measures for the relief of that condition instituted. When either albuminuria or hemoglobinuria occur, rest and low diet should be enjoined. During the attack, dry heat may be applied to the swellings. Morphine may be given if there is much pain. Tracheotomy may be necessary when the larynx is affected.

INTERMITTENT CLAUDICATION (INTERMITTENT LAMENESS)

Three forms of this affection have been described, *i. e.*: (1) Those due to an arteritis of the vessels supplying peripheral nerves; (2) those due to a vasomotor spasm of these arteries; (3) those due to a similar condition of spinal arteries.[1] The symptoms in general consist of cramps and weakness, usually in the legs, coming on after a moderate

[1] Arterial spasm causing transient loss of function may occur in cerebral vessels, even when arteriosclerosis is not present, pp. 106, 323, and 495.

amount of exertion. Pain, numbness, and other forms of paresthesia may also be present. The arms also may be affected (dysbasia or dyspraxia angiosclerotica intermittens). In the cases due to arteriosclerosis of the peripheral nerve vessels there is absence of pulsation in the posterior tibial and dorsalis pedis, and the vessels will feel rigid. This would not be so in those due to vasomotor spasm and the affected limbs would be pale. In the cord affection pulsation of the arteries above mentioned will be preserved; there will be no cyanosis or other vasomotor symptom, while the knee- and Achilles-jerks will be increased; a Babinski reflex and difficulty in holding the urine will be present. These symptoms, of course, being temporary and following muscular exertion.

The affection must be distinguished from *myasthenia gravis*. The presence of cramps, numbness, arteriosclerosis, vasomotor disturbances, and the absence of the characteristic symptoms of this disease (p. 267) enable the distinction to be made. The severe asthenia sometimes present in neurasthenia may also resemble it, as the weakness is more pronounced after exertion. Attention, however, to the history and careful examination will usually, without trouble, enable a diagnosis to be made.

Treatment.—The treatment is that of arteriosclerosis generally. Mercury has done good in the spinal cases. Nitroglycerin should be used if the blood tension is high. Potassium iodide is often of service.

PROGRESSIVE FACIAL HEMIATROPHY

Definition.—A rare disease, characterized by progressive wasting of one side of the face.

Etiology.—It usually develops between the ages of ten and twenty. Direct heredity has been traced to a few cases. The infectious fevers seem to act as exciting causes. It also occurs with disease of the fifth nerve.

Symptoms.—The disease first shows itself in patches and progresses gradually. The skin grows thinner, there is loss of pigment, and the hair falls out. At first the spots are white

in color, but as they grow larger they become of a yellowish tinge. As the skin and subcutaneous fat atrophy, pits or trough-like depressions are formed. The bones and, to a slight degree, the muscles are also involved. The sebaceous glands atrophy and their secretion becomes checked. Perspiration is either normal or increased. The atrophy may involve the tongue, hard and soft palate, the gums (with falling out of the teeth), and the uvula. Neuralgic pains and paresthesias may occur in the early stages of the disease. Rarely, there may be anesthesia. Spasm of the masseter, buccinator, and temporal muscles is a rare symptom. When the disease is fully developed the atrophied side is separated from the other by a deep groove; the skin has a roughened and puckered appearance; the hair of the head, beard, eyebrows, and lashes has fallen out in patches; the eyeball is sunk in and the palpebral fissure narrowed (Fig. 134).

FIG. 134

Case of facial hemiatrophy. (After Yonge.)

Pathology, Diagnosis, Prognosis, and Treatment.—There has been found a degenerative neuritis involving the branches of the trigeminus, most marked in the superior maxillary branch, and also atrophy of the ascending root of the fifth nerve (Mendel).

The disease might be *confounded* with congenital asymmetry and morphea. The former is noticed soon after birth, and remains stationary. The latter is considered in another place.

The disease does not shorten life, but its progress cannot be arrested by any known method of *treatment*. Tonics and galvanism may be tried.

FACIAL HEMIHYPERTROPHY

This is an extremely rare condition, in which one side of the face *progressively enlarges*. Care must be taken in this affection not to mistake the normal side for one that is atrophied; in other words, the condition may thus be mistaken for facial hemiatrophy.

SCLERODERMA

Definition.—This is a disease resulting in a diffuse or circumscribed atrophy and induration of the skin and subcutaneous tissue. When the disease is circumscribed it is commonly known as *morphea* or *Addison's keloid*.

Etiology.—The disease most commonly occurs in females. It may occur at any age. It may follow infectious diseases, exposure to extremes of temperature, trauma, mental and physical strain, and may also occur in those suffering from some other nervous disease.

Pathology.—The pathology of scleroderma is unknown. Changes in the bloodvessels of the skin and increase of connective tissue have been found. The thyroid has been found atrophied. Morphea may be confounded with facial hemiatrophy. All the tissues in the latter are effected primarily; in the former the subcutaneous tissues only atrophy from pressure.

Symptoms.—Pain in the joints or extremities may precede for a time the involvement of the skin. This is first noticed as a feeling of stiffness, most commonly felt at the back of the neck, the shoulders and arms, the face and scalp. The disease progresses slowly, and when the induration has reached its height the skin of the affected area is of leather-like thickness. It cannot be pinched up, nor can pitting be produced by pressure. If the skin over a point is involved, this becomes fixed as the skin becomes rigid. The face, if affected, loses all traces of expression; mastication may be difficult. Respiration may be interfered with if the skin of

the chest is involved. If there is a widespread involvement, the patient is in a state of more or less complete rigidity; in fact, as if he were encased in leather. The line of demarcation between the affected and the sound skin is not a sharp one. The color of the skin is usually white, but may be mottled or pigmented. The mucous membrane of the mouth, vagina, etc., may be affected. The secretions of the skin are diminished or absent. Vasomotor disturbances, as cyanosis of the legs and hands, are common. After the disease has lasted a time, atrophy of the subcutaneous tissues occurs and deformities result. That known as *sclerodactylie* consists of deformity, shortening, and atrophy of the fingers, with thickening and rigidity of the skin, which is either waxy in color or pigmented.

Morphea presents itself in the form of patches, bands, or streaks of various size. They are especially apt to occur on the breast and in the course of the fifth nerve. The patches are white or yellowish white in color, bordered by a pinkish zone. In general characteristics they resemble the patches in the diffuse form, but the rigidity may not be so marked. It is not infrequently complicated by Raynaud's disease.

Prognosis.—In the early stages recovery may occur. In cases of long standing this does not result. The disease itself does not produce death. It predisposes to affections such as rheumatism and pneumonia, which are apt to be fatal.

Treatment.—The patient should be warmly clad, and guarded against exposure. Tonics and nutrients, as arsenic and cod-liver oil, should be given. Warm baths, followed by frictions of oil, are valuable. Thyroid extract may be tried. Good results have been claimed from large doses of salol, gr. xv, three times daily.

SPONDYLOSE RHIZOMÉLIQUE

This disorder, also termed *ankylotic rigidity of the spine.* is probably a form of rheumatoid arthritis, but as one form in particular gives rise to symptoms of disorder of the

nervous system, a brief mention is here made. The disorder consists of progressive ankylosis of the intervertebral joints, causing rigidity of the spine. The process may begin in either the cervical or lumbar regions. The so-called von Bechterew type usually follows trauma or may be hereditary. In addition to spinal rigidity there are great pain, symptoms of nerve-root irritation, evidenced by shooting pains in the course of various spinal nerves, muscular atrophy, anesthesia, and possibly cord degeneration. In the Strümpell-Marie type, the nerve symptoms above mentioned are not so prominent, but may occur, and other joints, especially the hip and shoulder, are involved. In time the spine becomes completely rigid, with some kyphosis; the ribs become fixed, and hence breathing is abdominal. The nerve symptoms are due to pressure on the nerve roots.

Treatment.—The treatment is that of rheumatoid arthritis, *i. e.*, building up the general health, the use of arsenic, hypodermic injections of thiosinamine, hydrotherapy (hot compresses, hot baths), hot-air baths, counterirritation to the spine, massage, passive movements, and static or high-frequency electrical applications.

ACROMEGALY

Definition.—A disease characterized by overgrowth of certain parts, chiefly in the *bones* of the face and extremities.

Etiology.—It most frequently develops between the ages of twenty and forty-five. Trophic disturbances, causing enlargement of certain parts (as one limb), may occur in syringomyelia and tabes.

Morbid Anatomy and Pathology.—The disease is probably due to *hyperfunction* of the anterior lobes of the pituitary body. Altered secretions of other ductless glands, including the sexual, also have an influence. The pituitary is often the seat of tumor, either hyperplasia or adenoma, malignant adenoma, or sarcoma (p. 359). In other cases it is enlarged, but not the seat of any neoplasm. The thyroid is also usually abnormal, being either enlarged or atrophied. Other ductless

glands may be abnormal. Changes in the brain and involvement of cranial nerves may be present if marked enlargement of the gland exists. Enlargement of the internal organs may be found.

All of the bones are more or less affected, but the principal overgrowth is in the crests, tuberosities, and other bony prominences. Osteophytes are found. The outer parts of the bones are increased in density, while the spongy parts may be lighter than normal. There is also an overgrowth of subcutaneous connective tissue.

FIG. 135

Hand in acromegaly. (Osborne.)

Symptoms.—The symptoms of the disease develop gradually. Among the early symptoms may be noted a feeling of weakness, apathy, frontal headache, pain, and paresthesia in the extremities. In women, menstruation becomes irregular. There may be loss of sexual power and appetite. Disinclination for exertion and depression of spirits are also

early symptoms. Vasomotor symptoms, as hyperidrosis and polyuria, may also be noticed at this time. Soon it will be noticed that the hands, feet, and face are increasing in size, while the stature is growing less.

Fig 136

Case of acromegaly, showing facial expression. (Dercum.)

When the disease is developed, with some of the symptoms above mentioned, the hands will be found to be considerably and symmetrically enlarged, all the tissues being hypertrophied. The fingers are sausage-shaped, and the nails small, flat, and longitudinally striated. The general shape of the hand resembles that of a spade (Fig. 135).

The wrists are enlarged and the lines in the palms are deepened. The changes in the feet are similar. The great toe is often enlarged out of proportion to the rest of the foot, and the os calcis projects backward.

The lower jaw is markedly increased in size and projects forward. The lips are large, particularly the lower one. The nose is also enlarged, and often pigmented; and the tongue, palate, and alveolar processes are likewise hypertrophied. The supra-orbital arches are thickened, in some cases causing the eyes to appear deeply set; in others there is exophthalmos. The malar bones and zygoma stand out prominently. The ears are enlarged, the hair coarse and dry, and the beard scanty. The skin of the face is also dry, and may be pigmented. The expression is dull and rather sad (Fig. 136). The bones forming the thorax also increase in size, and the back becomes bent (kyphosis) (Fig. 137).

The external genitals in both sexes may be enlarged. The uterus is small, presenting the changes incident to senile involution. Contracted visual fields and hemianopsia, and varying degrees of optic neuritis and atrophy, have been reported. Nystagmus and paralysis of the ocular muscles may also occur. After the disease has lasted for some time, the muscles become soft and flabby.

The viscera are usually normal. Albumin may be found in the urine, which is increased in quantity. The thyroid may be enlarged. The voice is low pitched, resonant, and of a disagreeable intonation. Speech may be difficult, on account of the enlarged tongue.

Diagnosis.—Acromegaly must be differentiated from hypertrophic pulmonary arthropathy, leontiasis ossea, myxedema, adiposis dolorosa, elephantiasis, osteitis deformans, and local hypertrophies. The points of distinction between acromegaly and the first four diseases given will be mentioned under the description of those disorders.

In elephantiasis the thickening and increase in size are usually localized, and are confined to the skin and connective tissue. In osteitis deformans, or Paget's disease, the increase in bone tissue is most marked in the cranial bones, not those of the face; and the long bones which may

become curved and misshaped. The shape of the face is triangular, with the base upward, while in acromegaly it is egg-shaped, with the large end downward. The general involvement differentiates acromegaly from local hypertrophies.

FIG. 137

Case of acromegaly. (Osborne.)

Prognosis. — The disease is incurable, and terminates in death, either from exhaustion, suicide, or intercurrent disease. The duration is from ten to twenty years.

Treatment is symptomatic. If a pituitary tumor can be recognized it may be removed by surgical means. Extracts of thyroid and pituitary have been tried with not encouraging results.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Definition. — This affection is characterized by enlargement of the hands and feet, and of the ends of the long bones.

Etiology.—The disease is nearly always associated with some long-standing chronic disease, especially of the bronchi, lungs, or pleuræ. A few cases have apparently been due to syphilis. The disease has been thought to be due to the absorption of toxic substances, which exercise an irritant action upon the bones and articular structures.

Symptoms.—The hypertrophy of the long bones is confined to the lower three-fourths. The phalangeal, metacarpal, and metatarsal bones are also increased in size and club-shaped. The nails are large and curved over the ends of the phalanges. The face and head escape. There is often pain, and effusions into the joints occur.

Diagnosis.—Thayer tabulates the features which distinguish this disease from acromegaly as follows:

Acromegaly	Osteo-arthropathy
Subjective symptoms; headache; ocular disturbances. General nervous manifestations; sweating, polydipsia, etc.	Pain in joints of the extremities only.
Predominance of facial changes.	Absence of facial changes.
Changes mainly in soft parts.	No changes in soft parts.
Changes in the bones are generally the indication of an abnormal growth; and while inflammatory changes (periostitis) may be present, they are rare, and are usually limited to the points of muscular and tendinous attachment and the epiphyses, resulting in a general plumpness of the bone, with an exaggeration of the normal irregularities.	Characteristic periostitis, limited usually to the lower parts of the diaphyses of the long bones of the extremities, resulting in marked thickening and deformity of the bone.
Fingers flat and expanded laterally; nails relatively small.	Fingers clubbed; nails large and incurved.
Absence of joint symptoms.	Presence of joint symptoms.
Onset without apparent cause.	Secondary to some chronic, usually pulmonary affection.
Changes in the peripheral nerves are common.	Changes in the peripheral nerves are rare.
Tumor or disease of the pituitary body usual; goitre frequent.	Neither pituitary tumor nor goitre.

The **course** of the disease depends on that causing it. It does not end fatally of itself.

The **treatment** is that of the primary affection.

LEONTIASIS OSSEA

Leontiasis ossea is a rare affection, beginning frequently in early life, often as a result of trauma, in which there is hyperostosis of the cranial bones, and sometimes of those of the face. The soft tissues may also be affected. The restriction of the hypertrophy to the bones of the head and face suffices to distinguish the affection from acromegaly.

EXOPHTHALMIC GOITRE

Synonyms and Etiology.—This disease is also known as Parry's disease, Graves' disease, and Basedow's disease.

It is more frequent in women than in men. Most cases develop between the twentieth and thirtieth years. Worry, fright, and depressing emotions often precede the development of the disease. It may follow tonsillitis and other acute infections, and gastro-intestinal disturbances. It has occurred in several members of the same family.

Pathology and Morbid Anatomy.—The disease is due to disturbance of the functions of the thyroid gland, most likely an oversecretion. The amount of iodine found in the gland is much less than normal. The changes observed in it are characteristic, they consist of an increase of parenchyma and stroma with disappearance of the colloid. The epithelial cells are increased numerically, but grow in an atypical manner.

Symptoms.—The disease may be acute or chronic. The former is rare. It would be characterized by sudden development of the symptoms and a rapid course of the disease. In a case reported by J. H. Lloyd, death occurred in three days.

More commonly the onset is gradual and the course chronic. The prominent symptoms of the disease are tachycardia, exophthalmos, enlargement of the thyroid, and tremor.

An early symptom is the increased rapidity of the heart's action, which is extreme, reaching in some cases 160 or more. The action is usually regular. The area of cardiac pulsation is visibly increased, and the heart's action is strong and the sounds loud. The large arteries at the root of the neck can be seen to throb. The capillary pulse can be readily seen, as can also at times a venous pulse in the veins of the hand. On auscultation, murmurs are usually heard at the base and apex and over the sternum.

The exophthalmos follows the vascular disturbance. It may be unilateral, but usually is bilateral. The eyes protrude, so that the lids do not completely cover the sclerotics, showing a streak of white between the lid and cornea. The palpebral fissure is increased in width (Stellwag's sign). In some cases, when the eye is made to look down, the lid does not follow it, as in health (von Graefe's sign). The patients

wink less frequently than in health. The vision is normal, and there are usually no pupillary changes. Exophthalmos may sometimes be very slight in degree. The enlargement of the thyroid generally develops at about the same time as the exophthalmos. One lobe only or the entire gland may be affected. The gland pulsates, a thrill may be felt on palpation, and either a systolic or double murmur heard on auscultation.

The tremor is fine, and increased by exertion. Other symptoms common to the disease are anemia, loss of flesh, vomiting (which may be persistent), diarrhea, some elevation of temperature, physical weakness, and mental depression and irritability. There may also be flushes of heat and profuse perspiration. Pigmentation of the skin, urticaria, and edema may occur. Sugar or albumin may be found in the urine.

Diagnosis.—The prominent symptoms make the diagnosis clear. In the early stages the disease may be mistaken for neurasthenia. This is especially liable to happen when some of the cardinal symptoms, as exophthalmos and goitre, are not marked, as may sometimes be the case.

Prognosis.—The disease may cause death. The course is chronic, and it may last years. Cure may take place.

Treatment.—Rest and freedom from mental worry or excitement are important. For the tachycardia an ice-bag over the heart is most useful. The tincture of strophanthus or strychnine may also assist in reducing the rapidity of the heart. Belladonna, given until its constitutional effects are manifested, is of service. Phosphate of soda, in doses of 15 to 30 grains, three times daily, has given good results, and the use of phosphorus, associated with full feeding with milk, eggs, rice, meat, and fish, has been recommended. Good results have been reported from the exposure of the gland to the *x*-rays. Electricity may help, the cathode being placed at the back of the neck and the anode over the heart, and a constant current employed. Treatment by preparations of the serum obtained from the blood of animals, in whom the thyroid gland has been removed some time previously, has been employed with asserted success. This

may be obtained as thyroidectin, made by Parke, Davis & Co., which is given in doses of from 5 to 10 grains three times daily; and as antithyroidin made by Merck, of which 8 to 75 minims a day may be given. Rogers and Beebe[1] have advocated the use of a serum made from human thyroids. If anemia is present, iron must be given and the attacks of diarrhea treated with light diet, bismuth, and salol. In extreme cases part of the thyroid may be removed, or two or three of the thyroid arteries ligated.

MYXEDEMA

Definition.—A disease dependent upon atrophy of and consequent loss of function of the thyroid gland, and characterized by a myxedematous condition of the subdermal tissues and progressive mental failure.

Etiology.—It is more frequent in men than in women, and is commonly a disease of middle life. It has occurred after acute articular rheumatism, erysipelas, and persistent hemorrhages. Syphilis has apparently been the cause of some cases. It may follow exophthalmic goitre.

Pathology.—Symptoms similar to those described above are caused by the removal of the thyroid gland. In cases of idiopathic myxedema the thyroid is always found to be atrophied, and may be converted into a fibrous mass. To the loss of function of this gland the symptoms are due.

Symptoms.—Usually the onset and course of the disease are slow. Rarely the disease may be acute, as in the case described by Osler, in which there was a rather rapid development of the symptoms, associated with enlargement of the thyroid, these symptoms disappearing in a few months. There is a firm, inelastic swelling of the skin, which does not pit on pressure, most marked on the face and limbs. It is also dry and rough, there is absence of perspiration, and scaly particles of epithelium brush off. The hair is dry and brittle; and the nails brittle, striated, and either atrophied

[1] Jour. Amer. Med. Assoc., September 1, 1906.

or thickened. In the supraclavicular and axillary regions are tumor-like swellings. The physiognomy is characteristic, the features being coarse and broad, the lips thick, the nostril broad and thick and the mouth enlarged (Fig. 138). The color of the skin is yellowish white, and over each cheek, and sometimes on the nose, is a reddish patch.

Fig. 138

A cretin, aged about thirty-five years. (Philadelphia Hospital.)

There is slowness of thought and movement, the memory fails, the patient becomes irritable and suspicious, and finally hallucinations and delusions (with either maniacal or melancholic symptoms) may result. Speech is slow and difficult. The bodily temperature is below normal, 97° to 97.5°. A feeling of coldness is common. Albuminuria, and rarely glycosuria, may be present. Casts may also be found.

Diagnosis.—The fact that the swelling does not pit on pressure and is not influenced in location by gravitation distinguishes the disease from edema due to cardiac, kidney,

or liver disease. The features that distinguish it from adiposis dolorosa will be mentioned under that heading.

The **prognosis** is hopeful, but death may occur from the disease itself or from phthisis or renal or cerebral disease.

FIG. 139

A cretin. Same case as shown in Fig. 138.

Treatment.—The one remedy for this condition is thyroid gland, preferably in the form of the dried and powdered gland or of the glycerin extract. Of the former, a commencing dose is 1 grain three times daily, which is increased until 5 grains three times daily is being taken; or restlessness, delirium, and rapid pulse ensue, when the dose must be reduced. After the symptoms have disappeared, small doses

of the gland should be continued indefinitely. The patient should also be kept warm, and, if possible, move to a warm climate during the winter months.

CRETINISM

Definition.—This is a condition due to an absence or loss of function of the thyroid gland, which is either congenital or appears at any time before puberty.

Two **forms** are recognized, the sporadic and the endemic. In the *former* the gland may be congenitally absent. The condition may develop with goitre, or the gland may atrophy after one of the specific fevers.

Symptoms.—The child does not grow as it should, nor does it develop mentally. The tongue is large and hangs out of the mouth. The general appearance of the skin and features is as described under myxedema. The abdomen is swollen. A cretin of adult age may resemble in stature and mentality a child of two or three years (Figs. 138 and 139). Endemic cretinism is met with in Switzerland and in parts of Italy and France.

The **treatment** is similar to that advised for myxedema.

ADIPOSIS DOLOROSA

Under this title Dercum has described a disease occurring in adults, characterized by the deposition of fat in various parts of the body, which is first in the form of bunches or nodules, and later it is uncircumscribed and attended with pain, tenderness over nerve trunks, diminished cutaneous sensibility, and great muscular weakness (Fig. 140). There is frequently a history of either traumatism, alcoholism, or syphilis.

It differs from myxedema in the freedom from changes in the face, hands, and feet and the absence of marked mental symptoms, and the presence of pain. However, atrophy of the thyroid has been found in several instances.

It differs from the adiposis tuberosa simplex of Anders[1] in the absence of pain and tenderness.

FIG. 140

Adiposis dolorosa. (Dercum.)

The prognosis as regards cure is bad, the duration of life, however, is not interfered with. The administration of thyroid gland has been of benefit in some cases. The salicylates have some influence in relieving pain. Massage and baths may be tried.

[1] Amer. Jour. Med. Sci., 1908, cxxxv, 325.

MENTAL DISEASES

GENERAL CONSIDERATIONS

A PERSON suffering from mental disease or derangement of the higher faculties of the mind is commonly said to be *insane*. Scientifically speaking, however, all such are not insane, as will be seen.[1] Hence mental diseases will be treated under two heads —viz., *Insanity* and *Idiocy*.

Many **definitions of insanity** have been given by different authorities. None is entirely satisfactory. One of the simplest, and which covers the ground as well as any, is that of H. C. Wood: Insanity is a condition of mental aberration sufficiently intense to overthrow the normal relation of the individual to his own thoughts and acts, so that he is no longer able to control them through the will.

From the above definition it will be seen that, to exhibit symptoms of insanity, there must be a departure from a previous normal state. Therefore, to determine the sanity or insanity of an individual, we must discover if there has been a marked change in the former habits of the individual. According to this, *idiocy* and *imbecility* are not forms of insanity.

Idiocy, as defined by Ireland, is "mental deficiency or extreme stupidity, depending upon malnutrition or disease of the nervous centres, occurring either *before birth* or *before the evolution of the mental faculties in childhood*."

[1] Tic, neurasthenia, psychasthenia, the anxiety neurosis, and hysteria (pp. 430, 444, 454, and 455) are, properly speaking, mental diseases, but are not forms of insanity, as the patient recognizes the falsity of his morbid ideas, etc.

The word *imbecility* is used to denote a less decided degree of mental incapacity. The term *feeble-minded* is also used to denote these conditions.

Hence, according to this definition, an idiot or imbecile who from birth has been deficient mentally, is not, strictly speaking, insane. He might become so later; but such would have to be decided by the same tests as are applied to those who were previously mentally healthy—*i. e.*, a departure from a previous standard. Thus, if an idiot who had always been quiet suddenly became noisy and violent, or developed delusions and hallucinations, we could say that he has become insane.

Stigmata of Degeneration.—By *degeneration* we mean a marked and morbid deviation from the normal type. It is shown by a faulty or unbalanced development of the body, and by nervous and mental peculiarities constituting the so-called neuropathic state. These peculiarities are termed the *stigmata of degeneration.* The more important of these are as follows:

Anatomical Stigmata. *Cranial anomalies:* consisting of asymmetry of the cranium, microcephalus, peculiar shapes of the skull. Facial asymmetry, as excessive prognathism, large jaws.

Deformities of the palate and uvula: as the torus palatinus —*i. e.*, a projecting ridge or suture along the palatine suture; high, narrow, and asymmetrically arched palates.

Anomalies of the teeth: as macrodontism, microdontism, projecting teeth, badly placed or misplaced teeth, double row of teeth, teeth striated longitudinally or transversely.

Anomalies of the tongue and lips: as macroglossus, microglossus, and asymmetry of the two halves.

Anomalies of the eyes: as narrow palpebral fissure, muscular insufficiency, marked astigmatism, and nystagmus.

Anomalies of the ears: abnormally implanted ears—*i. e.*, they project too far or lie too closely, are placed too high or too low, too far forward or too far backward on the head. Excessively large ears—*i. e.*, absolutely too large or relatively too large. Ears which are too small. Ears that have a general ugly shape, that are markedly conchoidal in shape;

those in which the lobes are absent or adherent to the cheek; abnormalities in or absence of the development of the helix, antihelix, and other parts of the external ear.

Anomalies of the limbs: as congenital luxations, supernumerary fingers or toes, fusion of fingers or toes, missing fingers or toes, excessive relative length of the arms, etc.

Anomalies and malformations of the genital organs.

Anomalies of the skin: as excessive hairiness or absence of hair.

The peculiarities of the *palate* and *ear* are the most important.

Any of these stigmata singly may occur in apparently normal individuals; but when several are present in the same individual, or when combined with other peculiarities about to be mentioned, they are significant.

Physiological Stigmata.—Tremor, tics, nystagmus, hereditary defects in the muscular system leading to atrophies, excessive or defective sensibility of the cutaneous and special senses, defects in speech, perversions of the sexual and other instincts, a diminished power of resistance to nervous and emotional strains.

Mental Stigmata.—These include excessive egotism, emotionalism, eccentricities, etc.

Hematoma auris is due to an effusion of blood or bloody serum between the cartilage of the ear and its perichondrium. It causes a swelling of the cartilaginous portions of the ear, usually of the helix. It most often occurs in the chronic forms of insanity, especially paretic dementia and epileptic insanity, and may appear suddenly without obvious cause. It also occurs in the sane, due to traumatism.

INSANITY

General Considerations. — Some idea of the different symptoms occurring in mental diseases is essential.

Disturbances of the Emotions.—We speak of them being *exalted, depressed,* or *perverted.*

The **exaltation** is manifested by an unusual increase of the emotions which are normally "exalted," such as joy,

anger, etc., as in *mania*. *Depression* by a similar increase of the "depressive" emotions, as sorrow, fear, etc., as in *melancholia* (p. 539); or there may be alternation of these conditions, as in *circular insanity*.

A condition of true **emotional enfeeblement** or lethargy, in which stimuli which would naturally affect this or that emotion, make no impression, occurs in advanced dementia from any cause. This condition must be distinguished from melancholia, in which, as has already been stated, there is depression of the emotional nature.

Depression of the emotions may also be simulated by those who remain in a lethargic condition, the result of a *delusion* (p. 524), as when, for instance, the patient thinks that he has received a command from a higher power to remain in such a condition.

The **intellectual functions**, memory, reasoning, attention, etc., may be increased or lessened in power.

Increased intellectual function is rare, usually occurring in the early stages of mania.

Diminished intellectual function occurs in various functional and organic brain diseases (neurasthenia, brain tumor, etc.); but is most evident as either a primary symptom or sequelæ of mental disease. When acquired—*i. e.*, coming on in one who has been healthy mentally—it is known as *dementia*. The lack of mental power occurring in idiots and imbeciles is sometimes termed *amentia*. This latter term has also of late been applied to a group of mental disorders due to either exhaustion or some form of toxemia (p. 526), in which there is interference with associative memory, *i. e.*, incoherence and confusion.

The function usually first impaired is *memory*. Loss of memory is termed *amnesia;* this may be only for certain things or extending over a definite period. This is usually seen caused by illness or injury, as for instance the loss of memory which occurs during the delirium of acute illness. This is known as *retrograde amnesia*. It may be continuous so that the patient is permanently unable to store up memories. This is termed *anterograde amnesia*.

Hypermnesia is the term applied to an exaggerated degree of retentiveness. One form of interference with memory is that in which the patient does not recognize place or time; in other words, he has no knowledge of his environment and what pertains to it. This faculty, which depends principally on the power of recollection and redeveloping past impressions, is termed *orientation;* loss of it, *disorientation.*

Paramnesia is a disorder of memory in which events are apparently remembered that never happened. This gives rise to the symptom known as *confabulation* or *fabrication.* For instance the patient will describe in detail and with apparent correctness various events that have occurred either recently or some time back. Thus one who has been bedfast for days, will tell how he had been to the theatre the day before, describe the play, tell who he saw there, and so on. These falsifications of memory are termed *pseudo-reminiscences.* They are usually fleeting, new ones being detailed each day.

It must be remembered that *memory for recent events* may be *impaired,* when that for those long passed is preserved.

Loss of the power of fixing the attention is indicated by inability to concentrate the attention upon one subject for any length of time. The mind naturally wanders from one subject to another; but the normal individual can, more or less completely, exclude subjects foreign to that under consideration. In the individual suffering from failure of the mental powers, this power is much diminished or lost. It may also be due to inability to fix the attention for any length of time in one direction, the attention may wander rapidly from one thing to another, as is seen in mania (aprosexia).

Incoherence may be due either to weakness of attention or to heightened cerebral activity.

In the *former* it is due to inability to complete the mental act, the mind wandering to another subject before the previous train of thought is completed. (See also Amentia.)

When occurring in the *latter* condition, it is due to the excessive rapidity of the mental acts, thoughts rushing into the brain so fast that there is inability to translate them fully into words. This form is seen in maniacal states.

The attention of the patient may be so completely, absorbed by some thoughts, usually his delusions, that nothing else is noticed (*hyperprosexia*).

Disturbances of volitional action or will power are frequent. This may be diminished or lost, in which ordinary excitements produce to resultant action; even the most powerful inducements fail to influence the patient (decreased psychomotor activity), as in melancholia. The lower animal passions may, however, be active in these cases.

It may be excited—seen usually in maniacal and delirious states—in which there is great restlessness and desire to be on the move, talkativeness, desire to perform great things, which may be started, but never finished (increased psychomotor activity).

Another disorder of volition is **stereotypy,** which means the constant repetition of some particular movement or action without any definite aim or reason for it. We may have stereotypy of either attitude, movement or speech. The latter is also known as *verbigeration.*

When patients do just the opposite of that which they are requested to do, the condition is termed *negativism.* This is most commonly seen in katatonia. Without reason such patients refuse to do anything they are requested to do, usually doing the opposite. They resist attempts to force them. For instance, if asked to open the eyes they close them; they refuse to eat when food is set before them, but will steal it and eat ravenously at the first opportunity. This is not due to voluntary opposition, but to some derangement of will power.

Suggestibility is the opposite condition in which the actions of the patients are determined by impressions or suggestions derived from others. This is seen in *catalepsy* when the limbs remain in any position in which they are put, in *echolalia* and *echopraxia.* This condition is also known as *automatism.*

Stupor (p. 35) is a condition in which there is profound disturbance of consciousness (*clouding of consciousness*). There are, however, conditions of apparent stupor, as in the katatonic form of dementia præcox (p. 553), in which the

consciousness of the patient is not disturbed. This is known as *pseudostupor*.

Hallucinations and illusions may be symptoms of insanity. They are disorders of *perception*.

An **hallucination** may be defined as a perception, through any of the senses, without any external cause capable of producing it. In the order of frequency of their implication, the senses affected are sight, hearing, touch, smell, taste, sensations from internal organs, and motor. Some apparent hallucinations may be due to pathological or even physiological processes occurring in sensory end organs.

Illusions are closely related to hallucinations. They are due to an erroneous conception by the mind of an external object perceived by any of the senses, or, in other words, the perception of an object in characters which it does not possess.

Hallucinations and illusions may be due to other causes than mental unsoundness. They are only evidences of insanity when the patient believes that they are realities in spite of evidence to the contrary which may be obtained by the exercise of his other senses.

Morbid impulses or impulsions occur when the patient has an irresistible desire to do certain acts. Thus, to steal (kleptomania), to set fire to buildings (pyromania), to drink liquor (dipsomania), etc.

Compulsions consist of the performance of some action, as touching certain objects, rubbing the arms against the sides of the body, etc., or there may be a desire to commit an abhorrent act, as murder. If they are interfered with the patient feels uncomfortable, becomes weak, has vertigo, perspires excessively, all of which disappear when he performs the act (p. 454).

Clouding of Consciousness occurs when sensory stimulation is not perceived by consciousness. It may be normal, as when sounds which one is accustomed to hear are not perceived. In some forms of mental disease the patient may be so absorbed in his hallucinations or delusions that even marked sensory stimuli do not attract his attention; such a patient may by increasing the stimulus be made to notice

and interpret it correctly. It may be slight or the patient may be comatose (p. 35).

Delusions are false beliefs; they are not necessarily evidence of insanity.

An insane **delusion** may be defined to be "a faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being" (H. C. Wood). In other words, the essential feature of an insane hallucination or delusion is loss of the power to receive and weigh evidence.

The delusion may have for its basis an hallucination, may be due to some disordered bodily sensation, some trivial circumstance may give rise to it, or it may be self-engendered in the mind. For instance, the hearing of voices may give rise to the delusion of persecution, a real and persistent abdominal pain to the delusion of pregnancy, or the seeing of two persons conversing together that they are conspiring against the patient.

Delusions may be **classified** into expansive delusions, hypochondriacal delusions, delusions of persecution.

Expansive delusions usually relate either to the mental or physical qualities, wealth, or business prospects of the person who has them. Thus he is the strongest man in the world, has millions of money, is a great genius, etc. They are sometimes termed *delusions of grandeur*.

Hypochondriacal delusions relate to imaginary disease from which the patient thinks he suffers. Thus he thinks he has an incurable disease, or that, if he moves, his bones will break, etc. These delusions often are based upon some real but slight ailment. As has already been said, neurasthenics (p. 448) often exaggerate their symptoms and become hypochondriacal. Such patients, however, may be able to perform the usual duties of life, and hence should not be termed insane. But when these beliefs either dominate the actions of the patient or exist entirely in the imagination, they become evidences of insanity.

Delusions of persecution are usually associated with hallucinations. The patient believes himself to be the victim of persecution either by some unknown persons or agents; or

he may fix upon some certain person as the cause of his troubles, in which event he becomes a lunatic of most dangerous type.

Delusions must also be divided into **systematized** and **non-systematized delusions**: The *former* is one concerning which the patient *reasons*, and the reality of which he defends more or less logically. Thus, if a man asserts that a certain person is trying to poison him by means of noxious gases, and that it is being done because the patient had discovered a great secret concerning this person, he would have a *systematized delusion.*

But if the patient merely asserted that he was being poisoned without attempting to give reasons why it was so, it would be an *unsystematized delusion.*

When an idea dominates the actions of the patient and is regarded by him of more importance than circumstances should warrant is termed a *fixed idea.*

Imperative Conceptions or Obsessions.—Certain ideas, emotions, and impulses may arise in the mind of a patient which, to more or less degree, dominate his actions, but the falsity of which is recognized. Such ideas are known as *imperative conceptions.* The various morbid fears are spoken of on p. 454.

Autochthonous ideas are those which come into the patient's mind through what he believes to be the influence of others. *Flight of ideas* and *distractibility* are defined on p. 544.

Ganser's symptom is described on p. 476.

Morbid desires are perversions of natural appetites. For instance, the various forms of sexual perversion or the eating of filth, etc.

Abnormal development of the sexual passion is termed *satyriasis* in the male and *nymphomania* in the female.

Erotomania is a term applied to a condition in which a person conceives a strong attachment to one of the opposite sex, perhaps whom he has never seen, but in which there is no real sexual excitement.

Delirium.—When we have a perversion of mental processes, consisting of incoherent speech, hallucinations, illusions,

delusions, inability to fix the attention, restlessness, disorientation, etc., which is due to either organic brain disease, toxemias in infectious fevers, mineral poisons, alcohol, or inanition, we term the condition *delirium*. When due to toxemia and exhaustion it is called *confusion* (p. 528).

Classification.—A satisfactory classification of mental diseases is difficult, if not impossible, to make. There have been nearly as many classifications as there have been writers upon the subject. Such will continue to be the case so long as our knowledge is not founded upon a pathological basis, but not groups of clinical symptoms. They all have the disadvantage of overlapping more or less. The subject will here be treated in the following order:

Psychoses the result of intoxication and exhaustion	Acute. Chronic. Confusional insanity. Neurasthenia (p. 444). Psychasthenia (p. 454).
Psychoses connected with imperfect functioning of the thyroid gland	Myxedema, cretinism. Exophthalmic goitre.
Manic-depressive group	Depression. Excitement. Mixed.
Paranoia	
Dementia præcox	Hebephrenia. Katatonia. Paranoid forms.
Epochal insanity	Puerperal. Senile or involution psychoses.
Insanity connected with other neuroses	Epilepsy. Hysteria (p. 455). Traumatic neurosis.
Insanity connected with chronic physical diseases. Paralytic dementia.	
Dementia	Secondary. Organic.

PSYCHOSES DUE TO ACUTE INTOXICATION AND EXHAUSTION

These may be divided into the (1) deliria occurring with the infectious fevers; (2) acute delirium or delirium grave; (3) the subacute delirious states described as amentia, acute hallucinatory insanity, confusional insanity.

1. **Deliria Occurring with Infectious Fevers.**—The characteristics of the delirium occurring in febrile conditions have been described on page 525. Their occurrence associated with the other symptoms of the various infections, as typhoid fever, scarlatina, measles, pneumonia, etc., enable the diagnosis to be made. Patients delirious under these conditions, while technically insane, are not commonly so considered. Acute maniacal and other states, associated with excitement, may have to be excluded; in them, however, fever is absent or very slight, and the history of the development and course of the disease differs. It should be remembered that the conditions described in either Group II or III of this class (p. 526) may follow infectious diseases (see p. 218). The **treatment** is that of the disease causing it, hydrotherapy being of special value, if not otherwise contraindicated.

2. **Acute Delirium** (Delirium Grave).—Typhomania, hyperacute mania, acute delirious mania, collapse delirium, Bell's disease, phrenitis, etc., usually develops in neurotic persons who have been subjected to severe mental shocks, prolonged worry, trauma; during the period of defervescence after febrile diseases, after parturition, and surgical operations. The products of gastro-intestinal putrefaction may also have a causative influence. Females are more liable than males.

Symptoms.—Prodromes are usually of brief duration, and may be physical, consisting of anorexia, nausea, vomiting, and areas of hyperesthesia. Delirium soon appears, rapidly becoming more and more violent; hallucinations, at first of a fantastic and bizarre character, soon become of a terrifying nature, and the patient becomes frenzied in his efforts to escape from them. Disorientation is soon complete, and friends and relatives are not recognized. Motor restlessness is extreme and insomnia profound. The pulse is rapid and the temperature ranges from 100° to 104° or more. Food is refused, and the tongue is dry and coated; gastro-intestinal symptoms may be pronounced. Most cases, in the course of a few days, sink into a comatose state and die from exhaustion. Remissions may occur, and a few may recover who afterward pass into a state of more or less complete dementia.

Diagnosis.—In ordinary acute mania the symptoms are not so violent; there is usually a more or less period of preceding depression, and fever is either absent or very slight. The symptoms also may continue for a long time. In delirium tremens there is a history of prolonged alcoholic excess, the presence of tremor and absence of the extremely violent delirium and high temperature described above. Meningitis may be distinguished by its physical symptoms (p. 218). The history of the onset and course of the disease with the physical symptoms present would distinguish the delirium occurring during the active stage of infectious febrile diseases.

Pathology.—Various organisms have been isolated from the blood and meninges in this affection, but nothing constantly present has as yet been discovered. Various forms of degeneration of cortical cells and nerve fibers have also been found, but nothing specific.

Treatment.—If possible, immersion in a warm bath should be carried out, the temperature being about 100°. While at first it may only be possible to keep the patient immersed for a few minutes, the period should be prolonged until he remains there for hours if possible, or wet packs may be used (p. 92). If evidences of marked toxemia are present, hypodermoclysis often proves useful. Otherwise the treatment consists of measures to stimulate the vital functions, and if the bath does not quiet sufficiently, veronal, hyoscine hydrobromate, etc., may be given. The diet should be concentrated, as raw eggs, milk, broths, etc., and given often in small quantities. If necessary, feeding by the nasal tube (p. 542) or nutritive enemata must be used.

3. **The subacute states of delirium and mental confusion,** described under the names amentia (Meynert), acute hallucinatory insanity, acute confusional insanity, hallucinatory confusion, and primary dementia.

Etiology.—This is practically the same as acute delirium (p. 527). Females are also more liable to be attacked than males. The disorder usually occurs in individuals in the prime of life, but cases have been reported as occurring in children of nine years and in adults of fifty or sixty.

Symptoms.—There are two types: an asthenic, and one in which the symptoms are more violent. The former is the one usually termed *confusional insanity*, while the latter has been termed *hallucinatory confusion* and *delirious amentia*. In the asthenic form the patient is comparatively quiet and apathetic; incoherence is marked, he being unable to carry any line of thought to a conclusion. An example of the conversation of such a patient given by Spitzka is as follows: "I am, I—I don't know that—I—is dead—funerals are—How do you do?—met you on Boston steamer—this is London—I am sure of it—there are not so many now—look at that horse—it is alive—it is not a wooden horse—it looks so—I think it is—my poor father,"—and so on. Hallucinations, illusions, and delusions are present and may be of a pleasant nature one moment and horrifying the next, although they may not cause any corresponding emotional state. Disorientation is marked, and the patient often thinks that he is someone else or that someone is in bed with him. Memory is markedly impaired, and the attention cannot be held for any length of time. Tremor of the facial muscles may be present, the temperature may be either slightly elevated or subnormal, the pulse small and rapid, weight is lost, and the reflexes increased.

In the latter or more excited form the symptoms resemble those of acute delirium, but are not so acute, and persist much longer.

Diagnosis.—The condition may be confounded with mania, the excited stages of dementia præcox, paresis, and chronic alcoholism. The first is most likely to be confounded. In mania, sensory impressions more easily deflect the patient's attention (distractibility). Apprehension is not pronounced, the memory is not impaired, the evidences of physical depression are also not so marked, while motor restlessness is apt to be greater, and marked temperature changes do not occur. In dementia præcox the confusion is less, while impulsiveness is greater; katatonic symptoms, while they may occur, are less pronounced in amentia, and a history of preceding infectious disease or other strain on the system is not a prominent feature in dementia præcox. The presence,

of the characteristic physical symptoms distinguish paresis, while the history will distinguish chronic alcoholism.

Uremia may cause similar symptoms of greater or less intensity which may come on acutely or be chronic in type, examination of the urine is therefore very important.

Prognosis.—Most cases recover, but some, especially of the asthenic type, may die in collapse. The duration may be from several weeks to several months. A gain of flesh is usually a favorable sign. Sometimes more or less dementia, with delusions, may be permanent.

Treatment consists of hydrotherapeutic measures, as warm baths or packs for excitement, sedatives if necessary; hypodermoclysis if collapse threaten, with tonics and forced feeding.

PSYCHOSES DUE TO CHRONIC INTOXICATION

The principal cause of these psychoses is alcohol, but the prolonged use of other drugs may also cause mental symptoms, notably ether, paraldehyde, bromides, opium, and cocaine. Chronic lead poisoning may also cause symptoms belonging to this class. It is important to remember that unusual forms of alcohol, as cologne water, bay rum, Jamaica ginger, etc., may be taken, especially by women. Alcohol causes several different forms of mental disturbance, the most common of which is delirium tremens. Other forms are the acute alcoholic paranoia or alcoholic hallucinosis of Wernicke and Bonhoeffer, conditions simulating paranoia and forms of dementia, notably paresis, and Korsakow's psychosis or disease.

DELIRIUM TREMENS

Symptoms.—The symptoms may develop after a sudden cessation of the use of stimulants or come on during a prolonged debauch. A shock to the system, such as trauma, the development of some acute disease, surgical operations,

and mental strain, is prone to act as an exciting cause in heavy and habitual drinkers.

The mental symptoms in *mild cases* are: mental confusion, depression of spirits, forebodings of evil, and a sense of terror with possibly hallucinations at night.

In *severe attacks*, hallucinations, especially of sight, are marked and constant. These are always of a horrible and terrifying nature. Thus the patient sees disgusting objects. While these patients may be violent, it is due to terror and an effort to defend themselves. Confabulation and disorientation are prominent symptoms in severe cases. The physical symptoms are characteristic, and consist of marked tremor, physical weakness, insomnia, loss of appetite, and often weakened heart action. Pneumonia is apt to occur. A sluggish reaction of the pupil to light may be present and albuminuria will be found in many cases.

Diagnosis and Prognosis.—The peculiar character of the hallucinations, combined with the tremor, is characteristic.

While most of these cases recover, in broken-down individuals, or in those whose hearts are weak, death often occurs. Pneumonia is a very dangerous complication. Alcoholic cerebral edema (p. 228) may occur. Some cases may pass into the chronic condition described below.

Treatment.—In treating such a case the first indication is the elimination of the poison. If there has been alcohol taken recently and the patient is in good physical condition, an emetic may be given. In all cases a purgative, preferably calomel, is essential, and measures to increase action of the skin and kidneys are of service.

It is essential to induce sleep. To this end hot packs, the bromides combined with chloral, chloral and morphine, paraldehyde, and other hypnotics may be used. Large doses of these remedies are often essential, but when used their effects must be carefully watched. If during the day there is much restlessness and nervous excitement, bromides combined with a bitter tonic and capsicum should be given at regular intervals.

In the majority of cases the use of alcohol in all forms should be stopped at once; but in old people, or in those

whose circulation is very feeble, it may be necessary to administer it and gradually reduce the amount. The use of full doses of strychnine is advisable. If the heart is very weak, digitalis should also be given.

Nutritious and easily digested food, consisting of milk, strong broths, highly seasoned with capsicum, soft-boiled eggs, etc., must be given as freely as can be digested.

If the patient is very violent and cannot be controlled, it may be necessary to restrain him by securing his person to the bed by straps or otherwise. When convalescence begins a prolonged sojourn in the country or seashore or an ocean voyage, with freedom from worry or anxiety, is advisable.

ACUTE ALCOHOLIC HALLUCINOSIS

The **symptoms** of this condition are very similar to those of delirium tremens. The differences are the predominance of auditory hallucinations instead of visual, and an attempt on the part of the patient to explain the various hallucinations and delusions which appear in the course of the disease. Hallucinations of smell and taste are not infrequent, but those of sight are rare. Delusions of persecution are prominent, especially those of marital infidelity, and the patient is unduly suspicious. Confabulation is not apt to be present and disorientation is not pronounced. Incoherence also is not marked, as in delirium tremens. Ideas of grandeur are sometimes present. The psychosis can be distinguished from paranoia by the rapid development of the systematized delusions as opposed to their slow evolution in the latter disease.

Prognosis is usually favorable, recovery taking place in from six weeks to three months. Some mental defect may be permanent (see below). It has been said that the nearer the symptoms resemble those of delirium tremens the better the prognosis.

Treatment is similar to that of delirium tremens.

PARANOIAC AND DEMENTED STATES

These may follow either delirium tremens or acute hallucinosis, or be primary. In the first variety, the patient recovers from the attack, with the exception that one or more persistent delusions remain. These are usually of a persecutory nature, and are especially apt to be directed toward the patient's family, particularly the fidelity of his wife. Violent outbreaks are apt to occur. More or less mental impairment usually coexists. Excessive sexual desire is frequently present, to become entirely lost later, which fact is often the starting point of the delusions of jealousy present. At times depression may be prominent, at other times exaltation. Hallucinations are not prominent. As time goes on dementia becomes more and more pronounced. These cases, in view of the existence of more or less systematized delusions of persecution, have been termed by some alcoholic paranoia. The delusions often appear so reasonable and likely to be true that only investigation will disprove their falsity. Similar delusions may also occur in senile insanity. The lack of a permanent systematization of the delusion and the history and symptoms of chronic alcoholism distinguish it from true paranoia. Sometimes they develop the peculiar complacent condition characteristic of paresis. Tremor, incoördination, and disturbances of speech also may develop, causing a still more pronounced simulation of true paresis. Pupillary rigidity may occur. Remissions in the alcoholic form may persist for years, while in paresis they usually do not last longer than a few months. In true paresis examination of the cerebrospinal fluid will show the characteristic conditions (p. 417). The symptoms of the primary form are similar, the only difference being their gradual development without preceding more acute disturbances.

Prognosis and Treatment.—Under proper conditions, *i. e.*, abstinence from alcohol, and a life free from strain, much improvement may occur. Some mental impairment, however, usually remains. The character of the delusions in many of these cases makes them dangerous, and they should

be placed under restraint. In addition to abstinence from alcohol, and a quiet life, large doses of strychnine and hydrotherapeutic measures are often of service. Special attention to the kidneys, bowels, and circulation is essential, as these organs are apt to be diseased in alcoholics.

Korsakow's disease, or polyneuritic psychosis, is usually associated with alcoholic neuritis (p. 126), but may possibly occur when alcohol is not a factor, other forms of toxemia, as typhoid fever, tuberculosis, uremia, diabetes, hepatic disease, lead and arsenic, being causes.

Pathology.—In addition to the changes due to neuritis (p. 119), various evidences of degeneration of cortical cells increase in the neuroglia, and some disappearance of cortical fibers may be present.

Symptoms.—The characteristic symptoms are incoherence, confusion, with a marked disposition to confabulate, and hence to narrate pseudoreminiscences; disorientation, hallucinations, and delusions which resemble in character those present in other forms of delirium. There may be either exaltation or depression, and memory is impaired. Such symptoms occurring usually with evidences of multiple neuritis are characteristic.

Diagnosis.—Delirium tremens may be confounded, but does not present the tendency to confabulate; disorientation is not pronounced, and usually does not last a great length of time. The failure of memory is also not great. The same symptoms distinguish it from mania. In cases due to hepatic disturbance, biliary pigment has been found in the cerebrospinal fluid obtained by puncture, and the fluid was discolored.

Prognosis.—Death may occur, and those who survive are usually left more or less demented. Recovery may, however, take place.

Treatment consists, in addition to that of the neuritis (p. 132), of supporting the heart and other vital functions, attention to the bowels and kidneys, plenty of liquid diet, and, as convalescence occurs, a quiet life in the open air as much as possible. A prolonged sea voyage may be of benefit.

Paraldehyde.—Excessive use of this drug may cause symptoms very similar to those caused by alcohol. The odor upon the breath of the patient will usually give a clue to the source of trouble.

Opium.—The indiscriminate prescribing of opium or its alkaloids by physicians is one of the most frequent causes of the development of this habit. Prescriptions should never be given so that they can be renewed.

The mental symptoms of prolonged use of either opium or its alkaloid morphine are states of apprehensiveness and anxiety, undue suspiciousness, and hypersensitiveness. There is a marked tendency to lie and fabricate, so that any statement they make is of uncertain truth. They finally lose interest in everything except the procuring of the drug, to which end they resort to any expedient. Hallucinations and delusions sometimes develop, especially if alcohol, as it often is, is also used excessively. Homicidal and suicidal impulses also develop at times. The physical symptoms, especially after withdrawal of the drug, are characteristic, consisting of diarrhea, severe pain in abdomen and extremities, coldness of the surface and subjective chilly sensations, coated tongue, foul breath, insomnia, and tremor.

The treatment of these cases cannot be adequately discussed here. The principles to be observed are isolation, under the constant supervision of a nurse, gradual withdrawal of the drug, unless the patient is very robust, when it can sometimes be withdrawn suddenly—it can always be reduced one-half at once—the use either of large doses of bromides or hyoscine for several days, until more or less somnolence is caused, when they are gradually reduced, and strychnine, caffeine, and other tonics substituted. Capsicum and bismuth will usually control the pain and diarrhea. Food should be light and concentrated. Hot baths or packs are often useful, especially in the beginning of the treatment. The patient should remain under supervision for some time after he can get along comfortably without the drug, as relapses are frequent. The so-called

"Lambert treatment,"[1] which consists of purgation and the use of belladonna and hyoscyamus, is a useful plan.

Bromism.—Large doses of bromides produce a slowness of all mental processes. If very excessive, stupor may be caused. Sometimes maniacal conditions are produced, and in several instances intense melancholy with homicidal and suicidal impulses has occurred.[2] The pulse is weak, tongue coated, and breath foul. The treatment is withdrawal of the bromides and the use of strychnine and other cardiac tonics.

Cocaine.—The habitual use of cocaine is characterized by the rapid production of mental enfeeblement and physical weakness. Cocaine habitues are very apt to also use alcohol and morphine. It is taken either hypodermically or by inhalation.

Acute cocaine intoxication in one who is not a habitue is characterized by mental excitement, a feeling of well-being, and an increase of the pulse rate. The patient is talkative, and may be delirious. The symptoms usually in such cases resemble those of acute alcoholic intoxication. Drowsiness, and in some cases collapse, follows.

Chronic cocaine intoxication is characterized by lack of will power, failure of memory, incapacity for sustained mental work, talkativeness, which is often somewhat incoherent, and a tendency to write lengthy, meaningless letters, and what they term poetry. They are apt to be overenergetic, but accomplish nothing, become careless about their appearance, and neglect their business. They are also apt to be suspicious and at times irritable. Physically, they lose weight, become pale, the muscles are flabby and often show fibrillary twitchings. The appetite is poor, the pupils dilated, but react to light, and the heart's action is weak.

These cases may develop at any time an acute psychosis resembling *acute alcoholic hallucinosis*, in which there are hallucinations, especially of sight and hearing. Of the former variety, black specks floating about, which are mistaken

[1] Jour. Amer. Med. Assoc., September 25, 1909, p. 985; ibid., February 18, 1911, p. 503.

[2] S. Weir Mitchell, Univ. Med. Mag., June, 1896, p. 661.

for insects, are characteristic. A sensation of foreign bodies being under the skin is also common. They are exceedingly suspicious, and often irritable and given to outbursts of anger. Delusions of marital infidelity are frequent, and when such become present they are prone to commit assaults and murder. Sometimes, however, suicide is attempted. The acute symptoms usually disappear in a few weeks after withdrawal of the drug; delusions may, however, remain.

The symptoms develop more rapidly than the similar condition due to alcohol, and they are more severe. Delusions of infidelity appear sooner. The sensation of objects being under the skin is characteristic.

The **prognosis** for permanent cure of the habit is bad.

The **treatment** consists of rapid withdrawal of the drug, which may be followed by shortness of breath, palpitation of the heart, insomnia, and at times a tendency to faintness. The use of caffeine and strychnine will relieve these. Insomnia is best treated with warm baths or packs; hypnotics can also be used if necessary; nutritious and concentrated diet is essential. The patient should remain in an institution for a long time after he is apparently cured. If morphine is also used, cocaine should be withdrawn first. The reckless prescribing of these drugs by physicians is responsible for many cases of the habit. Patients should never be given prescriptions containing them that can be renewed; neither should they know what they are taking.

Lead.—Chronic lead poisoning sometimes produces a mental condition called lead or saturnine encephalopathy. The mental symptoms are preceded by the usual physical symptoms of lead poisoning. Headache then develops, followed by insomnia, hallucinations, and sometimes violent delirium. Coma may or may not occur. Lead may also cause symptoms simulating paresis.

The **treatment** consists of the usual measures to eliminate the lead. Sedatives and measures to support the vital functions if necessary.

Pellagra.—Depression and confusional states may occur in this disease. The intestinal disturbances and appearance of the skin should enable the diagnosis to be made, especially when they occur in localities where pellagra is endemic.

PSYCHOSES CONNECTED WITH IMPERFECT FUNCTION OF THE THYROID GLAND

These cases consist of the mental disturbances present in myxedema (p. 512), cretinism (p. 515), and exophthalmic goitre (p. 509).

MANIC-DEPRESSIVE INSANITY

Kraepelin was the first to show that the symptom groups which for years have been known as melancholia and mania are more or less related—that is, the same patient may have had at one time mania, at another melancholia,[1] while careful study of cases supposed to be one or the other of these symptom groups will show that symptoms of both are present, although those of one or the other usually predominate. In other cases there is a sequence characterized by mental health, depression, and excitement known as circular insanity. Kraepelin's definition is as follows: Manic-depressive insanity is characterized by the occurrence of groups of mental symptoms throughout the life of the individual, not leading to mental deterioration. These groups of symptoms are sufficiently well defined to be termed the manic, the depressive, and the mixed phases of the disease. The chief symptoms usually appearing in the manic phase are: psychomotor excitement, with presence of activity, flight of ideas, distractibility, and happy though unstable emotional attitude. In the depressive phase we expect to find psychomotor retardation, absence of spontaneous activity, dearth of ideas, and depressed emotional attitude; while the symptoms of the mixed phase consist of various combinations of the symptoms characteristic of both the manic and depressive phases.

Etiology.—The most active factor in the etiology is the inheritance of a nervous system that is feeble, unstable,

[1] This does not include the melancholia of involution (p. 561).

and possesses deficient powers of recuperation. Hence the existence of various forms of nervous disorder in the family is frequent, and various stigmata of degeneration are often present in the patient. In such individuals any unusual strain upon the mind or body may cause an attack. Hence puberty, the climacteric, pregnancy, prolonged physical or mental exertion; mental shocks, either good or bad; acute and chronic diseases may act as exciting causes. These are the most frequent of all forms of mental disorder (70 to 80 per cent.). It is more frequent in women than in men. The first attack, in two-thirds of the cases, occurs before twenty-five years of age, but extremes from ten years to seventy have been noted. There are no demonstrable lesions.

THE DEPRESSIVE FORM OR MELANCHOLIA

Definition.—Melancholia may be defined as "a disorder characterized by a feeling of misery which is in excess of what is justified by the circumstances in which the individual is placed."[1]

Symptoms.—**Simple melancholia,** or that in which there is a prolonged period of mental depression without delusions, will be first considered.

The symptoms come on gradually: at first insomnia and pain or sense of pressure at the vertex or occiput, loss of appetite, constipation, failure of digestion, irritability, and some depression of spirits will be complained of. As the condition progresses the symptoms continue, while the patient becomes more and more depressed. He loses all interest in his business and family; has forebodings of all sorts of future trouble, and dwells upon his past errors of life and deficiencies. When aroused a coherent conversation can be maintained and a clear account of the symptoms given. He frequently expresses a desire to die. Food is either refused or only sparingly taken by him when forced. The expression of the face is that of abject misery, and the

[1] Tuke's Dictionary of Psychological Medicine.

eye is expressionless. The temperature is normal or subnormal. The tongue is flabby and frequently coated, the bowels constipated, and the urine scanty, high colored, and often full of urates. Anemia is often present, and in women menstruation is scanty or absent. Some loss of weight may occur.

Melancholia with Delusions and Agitation (Melancholia Agitata).—If the symptoms described above do not ameliorate, they usually become more aggravated. Hallucinations and delusions appear; for instance, voices are heard reproaching him for the commission of all sorts of crimes; he states that he is doomed to eternal punishment, or that dire misfortune will overtake himself and family, or already has done so. Insomnia and refusal of food become more marked; the patient is restless, may walk continually to and fro wringing the hands and weeping and wailing. Suicidal impulses are marked, and also in some cases are homicidal ones. Such patients often injure their persons by picking at parts of the body until the skin is worn away, pulling hair from the head, or, in extreme cases, may actually mutilate themselves. The extremities are cold, possibly congested, and all the bodily functions are in abeyance.

Melancholia with Delusions and Stupor (Melancholia Attonita).—Some cases, instead of passing from the simple into the agitated form, pass into a stuporous condition, or stupor may follow agitation. This may occur comparatively suddenly, and is due to the domination of delusions and to more or less suspension of the will-power. Delusions such as "they would go to pieces if moved," "that the world had come to an end," are types. During the height of the disease it is impossible to ascertain the nature of the delusions. The patient refuses to talk, and sits absolutely quiet with the head down. The expression is either anxious or terrified, and the eyes fixed. If made to stand, the position is maintained until he is made to change it. When an attempt is made to cause him to change the position, it causes irritation and may be resisted. Rigidity of the muscles is common. Some cases are cataleptic. Food is either not taken at all or only when placed in the mouth, when it may

be swallowed. Urine and feces, when passed, are done so involuntarily. The temperature is subnormal, and the extremites cold and cyanotic. There is marked loss of weight. In the above conditions the memory is not affected.

Diagnosis.—Simple melancholia in its early stages may be confounded with *neurasthenia*—in fact, neurasthenia may precede the development of the melancholia. When the depression becomes so great as to dominate the life of the patient, the latter may be said to supervene.

Hypochondria also resembles melancholia, and may lead to it. The hypochondriac is constantly looking for symptoms of disease, and exaggerates any abnormal sensation which he may have; but so long as he is not affected in his relation to his business and family affairs, and has no actual delusions, he cannot be said to be insane. Hypochondriacal delusions, on the other hand, are often present in cases of melancholia. For the distinction between this and the melancholia of involution see page 562. The differences between the stuporous form and the hebephrenic and katatonic forms of *dementia præcox* are given on page 558.

Cases of *general paresis* in which there is depression may be mistaken for melancholia. The finding of the physical symptoms of the former disease (p. 416) will enable the distinction to be made.

Paranoia, with depressive and persecutory hallucinations and delusions, is distinguished by the facts that the delusions are systematized and that the patient thinks he is persecuted wrongfully; while the victim of melancholia believes that he deserves his sufferings.

Prognosis.—This is fairly good, about 60 per cent. of the cases recovering, although, as has been said (p. 538), they are liable to have other attacks in the future either of this or the manic form. It is best in the simple, worse in the stuporous form. The average duration is from nine to twelve months; but recovery may occur either sooner or later. It has occurred after the condition has existed for years. Cases which do not recover die from exhaustion or end in terminal dementia (p. 569).

Treatment.—The first question that often arises is, Should the patient be treated at home or in a hospital? If the case is simple and the patient possesses means to enable him to carry out the measures essential, he may be treated at home. In severe cases with delusions, or when the financial resources are not ample, a hospital devoted to the treatment of mental diseases is the most suitable place. In the mildest cases the possibility of suicide must always be borne in mind and the patient closely watched.

A complete or partial rest cure is the preferable plan of treatment for mild cases. Rest in bed, say from 6 P.M. until noon of the next day, with an outdoor life for the remainder of the day, the ingestion of plenty of nutritious and digestible food, is an excellent plan. Massage and electricity (general faradization) should be employed when active exercise is not taken. The application of static sparks or high-frequency effluve is often of service in stuporous cases.

As some cases of melancholia appear to be due to an auto-intoxication, the bowels should be kept open by appropriate measures. In cases suspected of being so caused, high enemas of normal salt solution and intestinal antiseptics (salol or betanaphthol) may be given. Tonics of various kinds—strychnine, arsenic, mineral acids, and the glycerophosphates of sodium or calcium—should be prescribed.

For sleeplessness the warm bath or pack may be tried. When this fails, trional, gr. x to xx, in hot milk or soup, or veronal, gr. v to vij, are usually efficacious. When there is much agitation, opium given three times daily, beginning with a moderate dose and increasing if required, is often of service. Hydrobromate of hyoscine is also valuable.

If food is refused, the patient must be fed by introducing several times daily a soft-rubber catheter through the nose into the esophagus, and milk, eggs, and other concentrated food given through it. As soon as possible, mild, outdoor exercise (bicycling, golf, riding, etc.) should be encouraged. A sea trip is of benefit, but prolonged travel and its attendant excitement and fatigue should be avoided.

MANIC FORM OR MANIA

Definition.—Mania is a form of insanity characterized in its full development by mental exaltation and bodily excitement.[1]

Transitory maniacal symptoms may occur in paretic dementia, epileptic insanity, or may be due to toxemias, as lead, etc.

Symptoms.—Mania may be developed suddenly, but there is usually a **prodromal period** lasting from a few days to several months. These symptoms consist of depression of spirits, lassitude, lack of the power of fixing the attention, disinclination to work, loss of appetite, insomnia, constipation, etc.—in fact, the symptoms of a simple melancholia. The maniacal symptoms when they develop may be of various grades of severity and duration, hence we have the subacute and acute forms.

Subacute or Hypomania.—After the period of depression mentioned above the patient is noticed to be abnormally happy and gay. There is increased activity of all the physiological operations of the intellect. The patient may become much more active in his usual pursuits (occupation and otherwise) than is normally his wont (occupation delirium). Various projects—business, political, social, etc.—are devised, and as soon abandoned. The patient is loquacious, and his conversation may be witty and even brilliant. The memory is also more active than normal; and such patients are often able to remember events, to memorize poetry, etc., that in their normal condition would be impossible. A marked tendency to dissipation of all sorts often develops. There are also increased egotism and pugnaciousness; they may be said to go about with a "chip on their shoulders." Physically there are increased activity and restlessness; they always want to be on the "go," and seem incapable of experiencing fatigue. There are no delusions, and their actions and thoughts are coherent. In fact, in some cases it is only by noting the change from a previous normal condition that

[1] Tuke's Dictionary of Psychological Medicine.

the insanity of the patient is determined. This condition may be recovered from; or, after a greater or less period, passes into the stage of acute mania.

Acute Mania.—In this, the emotional excitement increases. Constantly shifting hallucinations, illusions, and sometimes delusions are present. The patient is noisy, and pours out continually a stream of incoherent threatenings, obscenities, prayers, and blasphemies. Ideas seem to crowd into the brain with such rapidity that he has not time to express them all; hence begins something new before he has finished the old (flight of ideas), and any slight incident at once suggests new combinations of words and acts (distractibility). Insomnia is marked. There is usually great sexual excitement. The patient is practically never quiet, rushing and jumping about his apartment, making grimaces and gestures; destroys his clothing and whatever else he can lay his hands upon. The appetite is voracious, but there is great loss of weight. The temperature is normal or very slightly increased. In very severe cases filth of all sorts is smeared upon the person, about the apartment, and even eaten. Alternating with these periods of excitement there may be brief periods of calm, even of depression (p. 538).

The symptoms in all cases of acute mania are not so violent as described above, but between it and the subacute form various degrees of severity occur. After recovery the patient remembers what has transpired.

Diagnosis.—In subacute and acute mania the history will generally make the diagnosis plain. It may, however, have to be distinguished from the *acute deliria* (pp. 527 and 528) and the excited stages of the hebephrenic and katatonic forms of *dementia præcox* (pp. 552 and 553).

Prognosis.—Cases of subacute mania usually recover, but are apt to have relapses. The average duration of acute mania is from three to six months, but may last longer. It may terminate either by recovery (70 per cent.), dementia, or death from exhaustion.

Improvement in the physical condition without coincident improvement in the mental is a bad prognostic sign, and indicates passage into the chronic or demented state.

Treatment. –Unless a patient has large means and can command the requisite attendance, removal to a hospital for the insane is the proper course to pursue. If treated at home, he should be constantly watched day and night; all furniture, excepting a mattress, which should be protected by a covering of canvas, removed from the room; and the patient dressed in a combination suit of similar material laced up the back. Mechanical appliances for restraint are rarely necessary, and are harmful.

Plenty of nutritious and easily digested food is essential. If refused by the patient, it should be administered as advised in cases of melancholia.

For the insomnia, prolonged warm baths (p. 92) or hot packs are often useful. If these fail, hyoscine hypodermically, trional, or paraldehyde may be tried. The bowels and kidneys must be kept active, and ferruginous and other tonics may be given. If great exhaustion is present, alcoholic stimulants are indicated. The treatment of these cases by absolute rest in bed has been very successful. Mabon has also obtained excellent results by keeping such patients continually outdoors.[1] If there is suspicion of previous syphilitic infection, specific treatment should be employed.

Mixed Forms.—Kraepelin has also described other forms:

1. **Irascible mania** in which there is a depressed emotional state, but the patient is very irritable, abusive, and fault-finding. There is usually, also, instability and physical restlessness.

2. **Depressed excitement,** in which there is restlessness out of proportion to the emotional despondency. They talk incessantly about the same thing, usually a hypochondriacal complaint, or that they have been abused in some way; at intervals they may be humorous and sarcastic, and become irritable and aggressive.

3. **Unproductive mania** is an excited state with a dearth of ideas instead of an increase as in the usual type. They are slow in understanding, a question having to be repeated several times; they do not pay attention, and give false

[1] New York Med. Jour., February 9, 1907, p. 241.

answers. The condition simulates imbecility, but at intervals they show that intelligence is not impaired. They are also elated emotionally and are incoherent. The emotional elation is shown by laughing loudly about nothing and the performance of mischievous tricks. Irritability is present. Periods of typical mania may occur.

4. **Manic stupor** is a depressive state, in which emotional elation takes the place of the usual despondency. They refuse to answer questions, take no interest in their surroundings, laugh without cause, may lie in bed rolled up in bedclothes, or dress themselves in a fantastic manner. Sometimes they are cataleptic. In the midst of this stuporous condition they suddenly show the ordinary symptoms of mania for a time, and then return to their former state. At times they may appear to be normal. Manic stupor may appear for brief periods in pronounced maniacal states (p. 544).

5. **Depression with a Flight of Ideas.**—These patients, while despondent, read a good deal, show some interest in their environment, but refuse to talk. When they do talk they state that their mind has been continually full of ideas that they had never thought of before. They often write voluminously about delusional ideas of persecution and fear.

6. **A Depressive State with Flight of Ideas and Emotional Elation.**—These patients are happy, show distractibility, irritability, and, if aroused by conversation, a flight of ideas (p. 544). At other times they are quiet.

Circular insanity occurs when a patient has regular periods in which he is normal for some time, then depressed, which is followed by excitement, or *vice versa* (Fig. 141), to be followed again by the normal state.

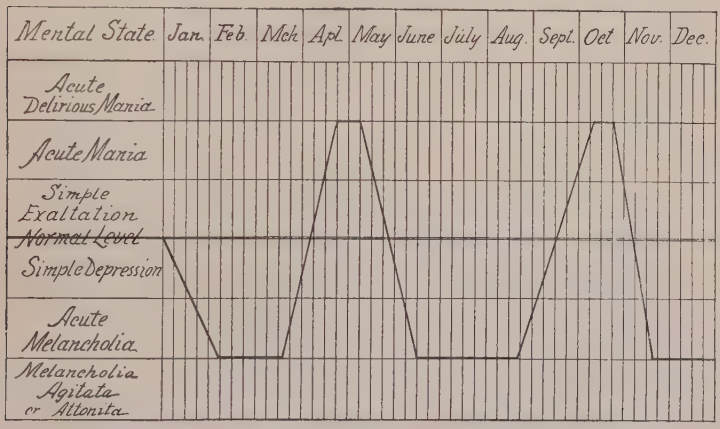
The **prognosis** in the mixed states as regards recovery is not good.

The **treatment** is that of the prevailing state (pp. 542 and 545).

Periodic or Recurrent Insanity.—This term is applied to attacks of either melancholia or mania, or of both, which recur at varying intervals of time, the patient being in a state of mental health between the attacks. Thus S. Weir

Mitchell has reported the case of a woman who, each March for sixteen years, has had an attack of melancholia lasting until the following September. The author has had under observation a girl who, for the last seven years, each December becomes maniacal, and remains so for from four to five months. This periodicity is not always a feature, the sane intervals differing in duration in the same patient.

FIG. 141



Scheme of course of disease in continuous circular insanity. (Church and Peterson.)

PARANOIA OR MONOMANIA

Synonyms and Definition.—This form of insanity, also sometimes termed progressive systematized insanity, has been defined to be one dependent upon original neurotic vices, accompanied by more or less distinctly systematized delusions, persistent, and without cyclical delusions (H. C. Wood).

The delusions may be of an ambitious nature, a form known as megalomania; or, what is more commonly the case, of a depressive nature, constituting the delusion of per-

secution. In some cases both forms may be combined. Such patients are often able to carry on business and other affairs of life without suspicion being directed to their condition; but close investigation will show that while such may be the case, they are to a more or less extent, dominated by their delusions.

Etiology.—As has been denoted by the definition, the great majority of these cases depend upon an hereditary neuropathy, or neurodegenerative taint. In some instances either prolonged worry, mental shock, or dissipation appears to act as an exciting cause. Some cases appear to be due to functional perversions of the cerebral faculties occurring in the course of dreams.

Symptoms.—In a large number of paranoiacs peculiarities have been noted before the actual outbreak of the disorder. *Mentally*, these consist of excessive egotism, irritability, suspiciousness, hypochondria, eccentricities in dress or manners, excessive timidity, imperative conceptions and impulses, agorophobia, mysophobia, or other similar peculiarities (p. 454).

Physically, some of the stigmata of degeneration are usually present, and a previous tendency to chorea, convulsions, and similar conditions will often have been present. Hallucinations, especially of hearing, frequently precede the development of delusions, which are then usually based upon them. The symptoms may become fully developed either early or late in life. Some cases never advance beyond the development of some of the peculiarities above mentioned. Usually the subject has a feeling that his merits are not appreciated as they deserve, begins to entertain suspicions that some person or persons are responsible for this state of affairs, and that some peculiar means, as the use of hypnotism, electricity, or poison in his food, is being used to destroy him. Then he finally settles upon the supposedly guilty person, who is often an agent of the government or in other high station.

With these symptoms hallucinations are usually present; he hears the voices of persons reviling or threatening him; he tastes the poison in his food or feels the poisonous gases

blowing over him. A conversation between two people on the street, a peculiar gesture, or other commonplace occurrences or remarks are regarded as directed against him and advanced as proofs of the correctness of his views.

Other patients think that they have made or are on the verge of making great discoveries; that they are great poets or have improved methods of government, which they are continually advancing (megalomania); or that they are beloved by some personage of higher station in life; or it may be that they love this person, whom they pursue with their attentions (erotomania). Then often, when they find that supposed merits are not appreciated, a cause is looked for and the delusion of persecution develops. At times they invoke the law to protect them, and, finding that it does not do so, it is taken into their own hands, and thus crimes are committed.

Another type is the *religious paranoiac*, who gives himself up to excessive piety and devotion, and either sees the Virgin Mary, Jesus Christ, or some of the saints, or hears them talk to him. Sexual perversions and masturbation are common in this form.

The above illustrate the usual forms in which this condition is manifested. Of course, different cases vary in the nature of their delusions and in the effects which their delusions have upon them.

At intervals, cerebral excitement, insomnia, or attacks of mania or melancholia may occur. Also, they often exhibit some of the many manifestations of hysteria, or are given to alcoholism and kindred vices.

Diagnosis.—In those cases in which the delusion is apparent, its mode of development, fixity, and systematized character make the diagnosis easy; but, on the other hand, cases occur in which the delusions are so conformable to what occurs in ordinary life that their recognition may be difficult. Thus, in a case detailed by Wood, a woman accused her husband of giving her syphilis, and it was only after prolonged investigation that it was discovered that the accusation was due to a delusion.

Evidence of a high degree of intellectual vigor in certain

directions is not against the existence of paranoia; great geniuses have been victims of it.

The intense egotism and suspicion which are present in nearly all victims of paranoia and evidences of a neuropathic diathesis may serve as useful hints in the investigation of such cases. It must be remembered that *chronic alcoholic intoxication* may cause symptoms simulating paranoia (p. 533). The paranoid forms of *dementia præcox* are differentiated on page 559. *Melancholia* may also simulate it (p. 541).

Prognosis.—It is doubtful if a paranoiac ever recovers. Remissions, however, may occur. The course of the disease is chronic, and usually ends after a long period in some mental enfeeblement.

Treatment.—The discipline and regular mode of living of a well-regulated asylum are beneficial. It may be, however, difficult to decide whether a man who is capable of conducting a large business or performing intellectual labor should be put there. While the insanity may be recognized by the alienist, it is often difficult to convince a judge or jury; and if such patients are committed, it is more hard to keep them there, as these are the cases which are continually applying for writs of habeas corpus, and make no end of trouble for the hospital authorities and those who have committed them. On the other hand, it must be remembered that a paranoiac with persecutory delusions is a most dangerous person to be at large.

The enjoining of a quiet, regular mode of living, freedom from care and worry, and an outdoor life will be of service as a prophylactic in those who exhibit the premonitory symptoms mentioned.

DEMENTIA PRÆCOX

(Schizophrenia)

According to Kraepelin this name should be applied to a group of cases which are characterized in common by a

pronounced tendency to mental deterioration of varying grades. He includes in this group the katatonia of Kahlbaum, cases formerly known as primary dementia, hebephrenia, and a group of cases simulating paranoia. These are classified into three groups:

1. The hebephrenic type.
2. The katatonic type.
3. The paranoid type.

It must be remembered, however, that these types may overlap more or less, and cases seen which embody symptoms of all forms, consequently often sharp lines cannot be drawn between them.

Etiology.—The disease is frequent, comprising from 14 to 30 per cent. of all admissions to hospitals for the insane. It is a disease of early life, most cases appearing before the twenty-fifth year. (The percentage varies in the different types.) It may, however, appear much later in life. The first and second types occur most frequently in males; the third is most frequent in females. A defective heredity is a prominent predisposing cause. Childbearing, acute illness, injuries to the head, and confinement in prisons seem to act as exciting causes. Some toxic factor, either an autointoxication or a disordered internal secretion, possibly of either ovary or testicle, has been thought by some to be the cause.

General Symptoms.—Many of the cases present some of the stigmata of degeneration, while some of them (33 per cent.) previous to the development of the disease have been bright. Others have presented peculiarities such as becoming delirious from slight provocation, convulsions in youth, susceptibility to alcohol, either the absence of sexual impulses or their early and unnatural development; eccentricities, precocious piety, seclusiveness, impulsiveness, moral instability, and weak-mindedness. Epileptiform or syncopal attacks may occur more or less frequently in the course of the disease; more rarely apoplectiform attacks, followed by a more or less permanent paralysis, occur sometimes as the first symptom of the trouble. Hysterical attacks also occur. Choreiform movements of the facial muscles, as wrinkling

of the eyebrows, distortion of the mouth, rolling the eyes, "making faces," smacking the lips, sniffing, grunting, etc., are very characteristic. The deep reflexes are usually increased, as is also the mechanical irritability of the muscles and nerves. The pupils are usually dilated, and may be unequal. Hypalgesia, vasomotor disturbances, excessive secretion of saliva and sweat, irregular and scanty menstruation, vascular disturbances, subnormal temperature, anemia, either anorexia or voracious appetite, gastro-intestinal disturbances, and, in the early stages, insomnia, and loss of weight may be all present more or less frequently.

Hebephrenic Form or Hebephrenia.—Definition.—"This form is characterized by the gradual or subacute development of a simple, more or less profound, mental deterioration" (Kraepelin). It represents about 58 per cent. of the cases of dementia præcox. Most cases develop previous to twenty-five years of age; many begin at the development of puberty, and it was formerly known as the insanity of pubescence.

Symptoms.—The onset is very gradual, and may be preceded by a period in which headache and insomnia are complained of. The symptoms may begin with a period of depression or sadness, during which suicidal attempts or efforts to self-mutilate may be made. The patients are anxious to talk about their sufferings to others, in which they differ from those suffering from melancholia; and at intervals during this period they may have spells of causeless laughter or make silly jokes, which symptoms are very characteristic. To awaken interest in themselves, they may simulate various disorders. After this period passes off they become restless, have no fixed purpose, trying first one business, then another. They often think that they have talents which are not appreciated as they should be. There is a marked tendency to use big words and foreign expressions, to be verbose or quote poetry.

Many of these patients previous to the onset of the symptoms are bright and in every way seem normal; in such the first symptoms will often be a lack of interest in things, they cease mental effort, lose ambition, and become the

opposite of what they have been. Gradually delusions and hallucinations, peculiarities of conduct, mannerisms, and irritability appear. Abortive forms of such cases may become tramps, criminals, or prostitutes.

Katatonic Form or Katatonia.—**Definition.**—This form, according to Kraepelin, is characterized by stuporous states, with negativism, hypersuggestibility, and uniform muscular tension; excited states, with stereotypy and impulsiveness; leading, in most cases, with or without remissions, to mental deterioration. It comprises about 18 per cent. of the total cases, and about 68 per cent. of those develop before the age of twenty-five years.

Symptoms.—The onset is usually subacute, and the symptoms resemble those of the hebephrenic form. During this period there may be some rise of temperature. During this period, in addition to the above, there is a tendency to assume peculiar attitudes, as laying with the arms extended in the form of a cross or holding the arms in awkward positions. There is also a tendency to perform rhythmical movements, as rolling the head from side to side, or continually expectorating. Sooner or later the characteristic symptoms occur of *katatonic stupor* and *katatonic excitement.*

The former is characterized by negativism (p. 522) and stupor; in the former they may refuse to speak or obey any command. When addressed they usually stare at some distant object, paying no attention to the questioner; they may, however, be made to sing. Every attempt to make them obey is resisted. Stupor is characterized by maintaining the same position for, in extreme cases, years. The eyes are rolled upward or stare into space, the hands tightly clenched, the muscles rigid, especially those of the arms, hands, face, and legs. The muscles of the mouth are often constricted and protruded forward in a peculiar way (Schnauzkrampf), food is refused, and they may soil themselves. Painful irritations will cause a response. If the patient does walk it is alone, in a slow, stiff manner, often on the toes or outer side of the feet, with body bent forward or backward.

The latter, which may precede or follow stupor, or occur

during its course, is characterized by *verbigeration*, or an uncontrollable and frequent repetition of the same word, sentence, or sound. This is a characteristic symptom of this disorder. Thus a patient of Peterson's would recite all day an infinite variety of rhymes of unintelligible words such as "tabies, gabies, habies, sabies, labies, mabies," etc., or "moccasins, noccasins, doccasins, crockasins," etc. At another period of the disease she would continually repeat the sentence: "Bring me home to my children in New York," accompanying this with theatrical gestures, by impressive actions, and stereotyped movements. The excitement may develop suddenly, and clothing is torn and furniture broken. They become noisy, very restless, and stereotypy is pronounced. They are apt to strike themselves or pound their heads against the wall. They may commit assaults and become filthy in their habits. Sexual excitement very often is increased. Other symptoms are the answering of questions in a foolish, irrelevant way, although it may have some reference to them, as, for instance, "Did you sleep well?" Answer: "It was a cold night." "How do you feel this morning?" Answer: "It is a nice morning," and so on (Ganser's syndrome, p. 476); the repetition of senseless expressions, and the use of sentences devoid of connection (*neologisms*). Many cases may develop no farther. In others, hallucinations, especially of hearing, develop. These are usually persecutory in nature, voices being heard making comments about their appearance, threats, etc. Hallucinations of sight may also occur, as visions of dead relatives, frightful accidents, crosses on the wall, etc. Hallucinations of smell also occur. Hypochondriacal and persecutory delusions develop in some cases, while in others they are of an expansive nature. Fabrications also become manifest, such as "they have been in Heaven;" "have just returned from a visit to Mars," etc. As the disease progresses these gradually become less and less prominent. While, at first, orientation is not impaired, it becomes so later in the course of the disease. Memory becomes gradually impaired and judgment is defective, so that he is unable to reason or perform mental work. They are usually dull and indifferent,

but may be self-centred and conceited. Religious enthusiasm may be great, while masturbation and peculiar sexual ideas are common. Mannerisms and peculiar actions and speech are often present.

Hypersuggestibility may be present in this stage, the patient obeying unreasonable commands, and also echolalia and echopraxia (p. 432). The states of negativism and hypersuggestibility may alternate, the patient passing from one to another in a few hours and back again.

The course of the disease is to dementia of a more or less profound type, with brief periods of excitement. Some apparently recover (13 per cent. according to Kraepelin), but are apt to have another attack later in life. Remissions lasting a greater or less period of time may occur.

Paranoid Forms. — **Definition.** — These differ from the hebephrenic and katatonic forms in that instead of the characteristic delusions becoming less marked as the disease progresses, they, together with the hallucinations present, are persistent through many years. In these forms also there is a more or less rapid mental deterioration, while consciousness remains clear. They comprise 22 per cent. of the total number of cases of dementia præcox, and consist of two groups:

The first, termed **dementia paranoides,** is characterized by the existence of numerous, persistent, incoherent, and variable delusions, which may be both of the persecutory and expansive type. There is also a moderate degree of excitement and a rapidly developing mental failure.

Symptoms.—The onset is preceded by a period in which there is headache, general discomfort, loss of energy, and more or less irritability. Symptoms of the hebephrenic type develop, to be soon followed by the development of a variety of delusions of the persecutory and hypochondriacal types. These are constantly changing. Hallucinations of hearing, in which they hear themselves called vile names, are present, and at times those of sight and smell also. They are agitated, restless, emotional, and irritable. Suicidal, homicidal, and other criminal impulses may occur. At other times they may become more or less exalted, and the delusions also

become of a more expansive type. They imagine that they are a distinguished personage, have remarkable powers, and so on. Intermingled with these, however, are absurd and peculiar delusions of persecution. They are apt to write copiously concerning these delusions, which descriptions are often more or less incoherent. Speech which at first is coherent and relevant later becomes the opposite. Orientation at first is not disturbed, but later is so, especially as regards time. They are unable to apply themselves to any sustained mental effort; egotism is often marked. Increased sexual excitement is common.

Physical symptoms in this form are not marked.

The symptoms of mental deterioration usually appear in about two months, and are progressive, without marked remissions, although it may be years before complete failure occurs. Transitory periods of excitement or depression occur, and there may also be periods of apparent improvement.

The **Second Group** consists of a group of cases characterized by "fantastic delusions, usually accompanied by numerous hallucinations, which are more coherently developed and expressed for a number of years, when they either become incomprehensible or disappear altogether, leaving the patient in a condition of moderate dementia" (Kraepelin).

Symptoms.—The first to appear are feelings of despondency with self-accusation. They often ascribe these feelings to previous masturbation. In time they become suspicious and more or less fixed, and systematized delusions of persecution appear. Hallucinations of hearing also appear at this time; these consist of hearing their supposed enemies revile them. Actual noises heard are misinterpreted to accord with the delusions. They often complain of poison being placed in their food. Hallucinations of sight may occur, but are rare. Various common sensations, as itching, muscular twitchings, headache, etc., are ascribed to the influence of their enemies, whom, they claim, use electricity, hypnotism, or some other means to accomplish these ends. They often think that their very thoughts are suggested and controlled by someone else or are known by the world, or that their organs have been removed to be replaced in an abnormal position, and

so on. At times more or less expansive delusions may also occur. In the course of several years the delusions become less and less systematized, and finally are incoherent and variable. Orientation is not affected for a long time. During the progress of the case the emotional attitude is first one of depression with anxiety and combativeness; later this gives way to a condition of elation and egotism. There may also be transitory attacks of either irritability or stupor. They often act in accordance with their delusions as the paranoiac does, applying to the police for protection, committing assaults upon their supposed tormentors, or otherwise endeavoring to protect themselves. Various mannerisms and eccentricities develop, and at times mild forms of negativism and stereotypy. In some of these cases the degree of dementia is very slight, and if it were not for the delusions the patient might be considered normal.

Diagnosis.—Many of the symptoms occurring in this disease also occur in other forms of mental disorder, and the diagnosis frequently has to be based on the previous history, the course of development, and the general disease picture presented.

The early stages of hebephrenia may resemble *neurasthenia*. The evidences of dementia, the general silliness, emotional apathy and lack of responsibility, and evidences of hallucinations, automatism, or stereotypy, that soon appear in the former, are never present in the latter. Cases occurring late in life may be mistaken for *paresis* in its early stages. The development of the characteristic physical signs of this disease will soon settle the diagnosis. Hallucinations, mannerisms, and silliness are not symptoms of paresis. In doubtful cases the changes found in the cerebrospinal fluid obtained by lumbar puncture (p. 417) speak for paresis. Acutely developing and excited cases of dementia præcox may be confounded with *confusional insanity* (p. 529). Negativism, stereotypy, catalepsy, eccentricities, silliness in answering questions, verbigeration, lack of interest, are not marked symptoms in the latter as they are in the former. In confusional insanity, also, the history of a previous acute illness or other exhausting condition (p. 528) will be obtained.

Beginning cases of katatonia, if preceded by an epileptiform seizure, may be mistaken for *epileptic insanity*. In the latter, orientation is more disturbed; there is more tendency to violence, assaults, and attempts to escape.

A history of a number of preceding convulsions would be in favor of epileptic insanity.

The depressive forms of *manic-depressive insanity* are apt to be confounded with the periods of depression that occur in hebephrenia and katatonia. The early appearance of many hallucinations, senseless delusions, especially ideas of physical influence, an emotional attitude which does not correspond to the depressive nature of the delusions, indifference to relatives and friends, non-performance of voluntary movements are characteristic of dementia præcox. In manic-depressive insanity lamentations are due to a feeling of grief and sadness in accord with the delusions present. The seeing of relatives is apt to increase the mental agitation; voluntary movements are performed, and are done so slowly; the resistance to passive movements is not so great as in the katatonic; and there is not the same persistence in returning to the original position after it has been disturbed.

Katatonic stupor is distinguished from manic stupor by the fact that in the latter apparent negativism is due to an irritability which leads to violence, more attention is paid to environment, orientation is not impaired, movements are purposive, and logical and not planless and impulsive, as in the former. Verbigeration does not occur in mania.

Katatonic excitement may have to be distinguished from the excited forms of manic-depressive insanity. In the former consciousness is less clouded; orientation is not so much impaired; verbigeration, stereotypy, and cataleptic states, common in katatonia, do not occur in mania. Distractibility, marked in mania, is not so in katatonia. The emotional attitude of the manic is exalted, often joking, while that of the katatonic is silly and indifferent. The movements of the katatonic are not purposive, while those of the manic are, and are dependent upon surroundings and

ideas. The motor restlessness of the katatonic occurs in a limited space, while that of the manic is only limited by his confines.

Excited stages of *paresis* may be confounded, but the physical symptoms present at once distinguish it.

Dementia præcox may have to be differentiated from *hysterical insanity*. It must be remembered that hysterical attacks may occur in the course of the former disease or may apparently precede its development. Hallucinations and delusions, if marked, are not apt to occur in hysteria. Hysterics also are sly, scheming, tyrannical to those caring for them, and capricious—symptoms not present in dementia præcox.

The paranoid forms resemble more or less pure *paranoia*. In the former the onset is more acute, the hallucinations and delusions developing much more rapidly, and they are less fixed and not so logically systematized. Silliness, disorientation, sexual excitement and general excitement often occurring in the former are not symptoms in the latter.

Imbecility may sometimes be confounded, and, in the absence of a history of the early life and development of the disease, may be difficult. It would then depend upon the appearance of some of the characteristic symptoms of dementia præcox, their variability and peculiar character of the silliness, hallucinations, and delusions.

Pathology.—This is probably a diseased condition of cortical cells, the exact nature of which has not been determined.[1]

Treatment.—Is best conducted in a well-regulated hospital for mental diseases. Life in the open air and physical labor seem to be beneficial. Excited states may be treated as in mania (p. 545).

EPOCHAL INSANITIES

This term is applied to mental derangements occurring during certain epochs in the patient's life, as during pregnancy, the climacteric, or in old age. As a rule the only

[1] Dunton, Amer. Jour. of Insanity, 1904.

special feature connected with them is the cause, they really being types of other forms of mental disease. They have, however, been so termed for many years, so that brief mention of them will be made.

Puerperal Insanity.—Definition.—This term has been applied to insanity occurring in women during the period of pregnancy or after delivery. It may appear within a few days or several weeks after confinement or during lactation. It is not a special form of insanity, but may manifest itself either in the form of confusional insanity, melancholia, or mania.

Etiology.—The worry and physical and mental strain which attend these states are, as has already been said, prominent causes of the development of mania, confusional insanity, and melancholia. Septic poisoning may also enter into the etiology.

Symptoms.—The symptoms in general are those of melancholia (p. 539), mania (p. 543), or confusional insanity (p. 528), more or less severe, as the case may be.

Prodromal symptoms are practically absent, the onset being rapid. There is often a marked tendency to commit suicide, to the infliction of self-injuries or mutilation, or to commit some act of violence on the child. The patients are also usually very suspicious of persons and food, and often refuse to take food. The temperature may or may not be elevated.

Prognosis.—While puerperal insanity is always a serious complication, if the temperature and pulse are not elevated and food is taken, the prognosis is favorable; but if the temperature and pulse are elevated, and if there develop refusal to take food, coated and dry tongue, much mental confusion and delirium, the outlook is more serious. Recovery, when it occurs, takes place usually in from three to five months.

Treatment.—When the physical condition of the patient will permit, removal to an asylum is the best procedure; but if the patient is very weak, treatment had better be conducted at home, at least until there is some improvement in this respect. In view of the nature of the symptoms, the

patient must be closely watched. Sleep should be induced by methods already detailed (p. 545), and plenty of digestible and nutritious food administered. If there is refusal to take it, forced feeding must be employed (p. 542). Tonics and stimulants should also be administered as required.

Senile Insanities or Psychoses of Involution. — All insanities occurring late in life are not of this type, as the acute deliria, manic-depressive insanity, paresis, etc., may develop at this time. There are, however, certain symptom groups that seem to be especially prone to occur at a period when physical deterioration, due to advancing years, is occurring. These have been named as above, and comprise melancholia of involution, presenile delusional insanity, and senile dementia. The two former occur earlier in life (fifty to sixty) than does senile dementia.

Melancholia of Involution. — **Definition.** — According to Kraepelin, this is characterized by "uniform despondency, with fear, various delusions of self-accusation, of persecution, and of a hypochondriacal nature, with moderate clouding of consciousness, leading, in the greater number of cases, after a prolonged course, to moderate mental deterioration."

Etiology.—The majority of cases develop between fifty and sixty, although it may occur as young as forty. It is most frequent in women in whom it seems to bear some relation to the climacterium (climacteric insanity). Mental and physical shocks and strain may act as exciting causes.

Pathology.—In addition to arteriosclerosis and the changes it produces in the different organs, evidences of degenerative changes in the nerve cells and an increase in the neuroglia have been found.

Symptoms.—The onset is usually gradual, there being a more or less prolonged period in which such prodromes as headache, insomnia, poor appetite, vertigo, general weakness, etc., exist. The main symptoms are briefly stated in the definition above given. In addition, hallucinations of sight and hearing may occur. They are coherent and may answer questions relevantly, and obey commands, but they are self-centred, constantly referring to their own miseries.

In some cases so-called "*nihilistic delusions*" are marked. These are characterized by the patient's claiming that nothing but themselves exists, even in some cases that they possess no organs. Sexual delusions, as they have been outraged, etc., may also occur. Restlessness may be a symptom, and the constant repetition of expressions indicative of lamentation is frequent. The facial expression shows the fear and anxiety that possesses the patient. Suicide is very apt to be attempted. The appetite is poor, insomnia pronounced, bowels constipated, tongue coated; the limbs may be cold, edematous, and cyanotic; pulse weak, and temperature subnormal. Signs of arteriosclerosis may be found.

Diagnosis.—The slow onset, greater anxiety, misery and restlessness, and the prolonged course as well as the age of the patient, distinguish it from the depressive form of manic-depressive insanity.

From the depression of katatonia it is distinguished by the absence of negativism and silliness, and by the relevance and coherency of conversation when it does not concern the delusions of the patient.

Prognosis.—This is not good, as only about 33 per cent. of the cases fully recover. Improvement occurs in others, with slight dementia, while about 25 per cent. become completely demented. Death may occur in a few years.

Treatment is similar to that of the depressive form of manic-depressive insanity (p. 542).

Presenile Delusional Insanity.—Etiology.—These cases are more rare; it is most frequent in women, and appears usually between the ages of fifty-five and sixty-five years. It may be hereditary.

Symptoms.—It is characterized by marked impairment of judgment, by unsystematized delusions of suspicion, especially of infidelity of the husband or wife; of being robbed or that they are being poisoned or mutilated. Hallucinations are not common, but those of hearing, in keeping with their delusions, may occur.

Memory for remote events is not impaired, as is also the case with consciousness, coherence of thought, and orienta-

tion. Suicide may be attempted, and they may become restless, irritable, and at times excited.

Diagnosis.—The unsystematized and unstable character of the delusions differentiates this condition from paranoia.

They differ from the rare cases of dementia præcox occurring late in life by the absence of katatonic symptoms and the fact that their restlessness and excitement depend upon their delusions, and are not senseless and without object.

Senile Dementia.—Etiology.—The condition rarely appears previous to the sixtieth year, but may occur earlier in those who have worked hard or been given to excesses. Traumatism, especially of the head, mental shocks, and infectious diseases may act as exciting causes. Degeneration of the bloodvessels and cardiac muscle incident to this period of life are usually found, together with small areas of cerebral softening, atrophy of the convolutions, dilatation of the ventricles, thickening and edema of the pia. Evidences of degeneration of the nerve cells, and increase of the neurogliar cells, which may also show evidences of degeneration and atrophy of the fiber tracts, are present. The spinal cord may also show evidences of atrophy of its cells and tracts.

Symptoms.—At first a lack of interest and ability to grasp details, and concentrate the attention, with failure of memory, are noticed. In the beginning recent events only are forgotten, while those which happened in the past are recollected. But as the disease progresses the loss becomes absolute. The patient forgets the names of his friends, where he lives, makes a statement in the course of conversation and in a moment or two repeats it. He frequently fabricates, to atone for the failure of memory. Such patients are usually irritable, and there is a tendency to restlessness, especially at night. They wander about the house, or, may be, away from it and lose their way. The symptoms may progress no farther, but if the disorder progresses the patient becomes unclean and negligent about his person; erotic tendencies also often become prominent; he falls in love with young women; may expose himself in public or attempt liberties with children; masturbation is also common. De-

lusions may occur, especially of persecution; they think they are being robbed or insulted. Hallucinations of like nature may be present. These symptoms may predominate without loss of orientation (senile delusional insanity). Owing to his lack of will and judgment, he may squander his property or make a foolish will. The symptoms may come on subacutely, with hallucinations of sight and hearing of a terrifying character; excitement, incoherence, disorientation, restlessness, and insomnia (senile delirium) or similar symptoms may occur for brief periods in the course of the more chronic condition above described.

In another form known as *presbyophrenia* the symptoms resemble somewhat Korsakow's psychosis (p. 534) in that they confabulate and are disoriented. The failure of memory is peculiar in that while they can enter into a prolonged conversation they forget almost immediately what they have been doing or what they have heard. They greet strangers as acquaintances, forget the ages and names of their children, refer to their dead parents as living. When cornered they either make some excuse for their lack of knowledge or fabricate. The absence of neuritic symptoms and the history and age of the patient distinguish this condition from Korsakow's psychosis.

Prognosis.—The course of this disorder is steadily onward, death usually occurring in from three to five years.

Treatment.—The treatment is hygienic and symptomatic. Opium, in cases attended by anxiety and excitement, is often of service. The erotic propensities and lack of will power existing in these patients must be borne in mind, and if they are not placed in a hospital, they should be closely watched. When wealthy they are often the victims of designing women, who induce them to marry them, or of relatives or other persons, who influence them in the disposition of their property.

INSANITIES CONNECTED WITH OTHER NEUROSES

Epileptic Insanity.—Definition and Varieties.—This, as its name implies, is the mental derangement which may occur

in *epileptics*. It has been estimated that 10 per cent. of all epileptics become insane. Various forms occur—*i. e.*, transitory, stuporous, and hallucinatory disorders, mania, melancholia, circular insanity, and finally dementia. Peterson makes the following classification: acute transitory epileptic insanity, chronic epileptic insanity, psychic degeneration of epileptics, and epileptic dementia. Epileptiform convulsions may also occur as symptoms in other psychoses, as paresis, chronic alcoholism, idiocy, dementia præcox, etc. These conditions must be excluded before a diagnosis of epileptic insanity can be made, the essential features of which are that the patient has been an epileptic previous to the oncoming of insanity, and that the attack, if transitory, occurs either before, after, or in place of a paroxysm. Many epileptics exhibit some evidences of mental deterioration, and are frequently irritable and hypochondriacal without being actually insane.

Acute Transitory Epileptic Insanity. — This may occur before, after, or in place of a convulsion. The duration of the attack may vary from a few hours to several days. The symptoms are various. More or less profound stupor, during which brief periods of excitement and confusion may occur, is a frequent form of this disorder. Consciousness may be entirely lost, but usually the state is one of partial consciousness. Associated with this there may be hallucinations, usually of a terrifying nature, but they may be pleasant; delirium, incoherence, delusions, often those of persecution, destructiveness, impulses to commit assaults, homicides, or suicide. Upon recovery there is either absolute loss of memory as regards everything that has taken place, or there is complete amnesia at first, with recollection of some events that occur as the condition is passing off. In this it differs from manic-depressive insanity, in which there is usually some, and often complete, recollection of what has occurred during the attack.

According to Peterson, the epileptic nature of such states, when the history is not known, may be suspected by the following characteristics: (1) Sudden onset and abrupt termination; (2) the terrifying or ecstatic nature of the

delusions and hallucinations; (3) disturbance of consciousness and stuporous conditions; (4) impulsive acts; (5) dream states; (6) amnesia.

Chronic Epileptic Insanity.—Epileptic automatism has already been described. (See Epilepsy.) The condition above described may take a chronic course, and resemble either chronic mania (p. 569) or the depressive or circular forms of manic-depressive insanity. Such occurring in an epileptic would be the distinguishing feature.

Psychic Degeneration.—The epileptic, in proportion to the frequency and severity of the seizures, suffers a gradual failure of mental power. Memory is impaired, ideation and expression are sluggish, the judgment is defective, delusions of persecution may occur. He is also apt to become irritable and brutal, and control over the passions is lost. During this period attacks such as above described (acute transitory insanity) may occur. The ultimate termination, if death does not occur before, is complete dementia. Of course this condition is incurable.

The **treatment** is that of epilepsy in general. The tendency to the commission of deeds of violence must be remembered, and it is well to commit epileptics subject to attacks of insanity to an asylum.

Hysterical Insanity.—Symptoms.—Some of the symptoms of hysteria may be so marked as to come under the definition of insanity. *Consciousness* may be more or less clouded either before, during, or after a convulsive seizure. This may last either a short or considerable time. Delirious states such as are described on pp. 457 and 458 are really a form of mental alienation. The Ganser syndrome (p. 476) occurs in hysteria. Memory of events occurring during the period of clouded consciousness is weakened. In some cases of what occurred in previous attacks is only recollected in subsequent ones (dual personality). Conditions of transitory excitement occurring as the result of jealousy, spite, or confinement in prison may occur; they are characterized by destructiveness, abusiveness, screaming, vile language, and sometimes even the smearing of the bodies with filth. These attacks usually pass off in a few days.

The **diagnosis** is based upon the ever-changing, shifting character of the symptoms associated with some of the stigmata of hysteria. It must be remembered that hysterical attacks may occur as episodes in the course of other psychoses as paresis, manic-depressive insanity, dementia præcox, etc. These are differentiated by finding other characteristic symptoms characteristic of them.

Restraint may sometimes be necessary; otherwise the **treatment** and **prognosis** is that of hysteria in general.

Traumatic Insanity.—Traumatism may appear to be the exciting cause of any of the psychoses. Confusional conditions (p. 528), which may terminate in dementia, probably are most frequent. As has been previously said (p. 530) alcohol is a predisposing cause. Paresis has been ascribed to injuries to the head; it is a doubtful cause, however, and syphilis and alcoholism must be excluded before such a diagnosis is made. It may, however, precipitate the attack in those predisposed. Dementia may also follow head injuries (p. 568). A peculiar mental state may occur in connection with the traumatic neuroses, which is described on page 479. Hysterical insanity, above described (p. 566), may also follow mental shock or physical injury.

INSANITY CONNECTED WITH CHRONIC PHYSICAL DISEASES

Mental derangements more or less frequently occur, associated with chronic physical ailments, notably tuberculosis, anemia, cardiac disease, syphilis (p. 410), gout, diabetes, nephritis, hepatic disease (p. 534), migraine (p. 106), chorea (p. 427). The derangement is not characteristic, but as in most cases it is probably due to an intoxication; the symptoms resemble those described as confusional insanity (p. 528) and Korsakow's psychosis (p. 534). In some cases, notably those of anemia and cardiac and vascular disease (p. 559), the symptoms may more nearly resemble those of dementia. The influence of syphilis in the causation of paresis is well known.

PARALYTIC DEMENTIA

This being an organic disease of the brain and spinal cord, and in which physical as well as mental symptoms are prominent, is more properly classified under diseases affecting the brain and cord on p. 414.

DEMENTIA

Definition.—Dementia is a term used to denote a more or less complete enfeeblement of the mental faculties of a mind that was once normal. The fact that the mental functions in a dement were once normal distinguishes dementia from idiocy and imbecility. The term is often used by the laity, and also sometimes by physicians, to denote any form of insanity. This is incorrect. It should be used only to denote those cases which come within the terms of the definition. A person afflicted with acute mania cannot properly be said to be demented.

Etiology and Varieties.—Dementia may be due to a number of different causes. To a more or less degree it may be due to disease of the brain, as tumor, meningitis, syphilis, epilepsy (p. 438), and cerebral apoplexy. It is also sometimes due to a prolonged toxemia, as from the effects of alcohol or lead (p. 418), and pellagra.

While some loss of mental vigor is a normal condition of old age, in some cases this loss goes beyond the normal limit and is associated with other peculiar features. Such a condition is known as *senile dementia* (p. 563).

Dementia also may be a sequel or termination of all insanities. It is then known as *secondary* or *terminal dementia*.

Loss of the mental faculties constitutes part of the symptoms of paresis, or *paralytic dementia*. Mental enfeeblement occurring as a consequence of brain tumor, meningitis, arteriosclerosis, apoplexy, or epilepsy, would be associated with the physical symptoms of these affections, which have been described. Dementia due to these causes would be

termed *organic dementia*. It shows more or less the symptoms to be described under the head of Terminal Dementia. Besides dementia, other symptoms of mental disease may occur in epileptics, which have been described under the head of Epileptic Insanity. Dementia due to syphilitic lesions will be described under differential diagnosis, between it and paresis, which it much resembles (p. 418).

Secondary or Terminal Dementia. — **Symptoms.** — The condition described as chronic mania may precede the development of secondary dementia, or the patient may pass into the demented from the acute condition. Chronic mania, so-called, is a form of terminal dementia following manic conditions; it is characterized by more or less fixed delusions, usually of an exalted character (this type has been also termed secondary paranoia). This is associated with loss of mental power, with at times attacks in which there is considerable excitement, restlessness, and other symptoms more or less pronounced of the acute manic condition (p. 544). In the mild cases there are loss of memory, some lack of judgment, and possibly some incoherence associated with delusions such as have been described as characteristic of chronic mania. If the condition is more pronounced, the patient has absolute loss of memory, judgment, extreme incoherence, and disorientation; the habits are filthy, soiling himself with urine and feces, and sometimes eating the latter. Masturbation is common. As children do, so they are likely to swallow anything which comes into their possession.

Paper, buttons, string, and all sorts of rubbish are collected and stored about the person. They are emotional, laughing or crying over trifles. Some patients are destructive and given to outbursts of excitement and passion; others are quiet, sitting in one place all day, having to be fed and tended as a little child. They are apt to become fat. The expression is vacant, either a silly smile or an angry frown being usually present. Such patients, of course, never recover, but they may live for years.

EXAMINATION OF THE INSANE

In examining a supposedly insane person two things should be determined: first, is he insane? and, second, if so, should he be treated in a hospital for the insane or at home?

When possible the physician should endeavor to ascertain **from the family** or elsewhere the habits and the previous mental and physical condition of the patient, and in what manner he has departed from that standard; the causes for such departure, and the existence of mental or nervous diseases in other members of the family or previously in the patient.

In **examining the patient** the condition of his physical health should be carefully investigated—viz., condition of circulation; presence of choreiform movements, of tremors; irregularities of the pupils; character of the speech; temperature; the reflexes; presence of emaciation; ataxia; motor paralysis; condition of the digestive organs, kidneys, heart, etc.

Particular attention should also be paid to the *facial expression,* whether apathetic, restless, frightened, etc.

Whether food is taken or not, and in what quantities, and whether insomnia exists or not.

By **conversation** with the patient we determine if the memory has failed both for past and recent events; whether incoherence and mental confusion exist; and learn the existence of hallucinations, delusions, simple depression, or exaltation. It may take some time and tact to determine these facts, and more than one visit may be necessary.

Removal to a Hospital.—Having determined that insanity exists, it is next necessary to decide as to the advisability of removal to a hospital.

If the patient has means, is not suicidal, homicidal, nor the victim of delusions that make him dangerous to himself and others, treatment at home may be effectual. When the patient is poor or the symptoms mentioned above are present, removal to a hospital is the best and safest plan both for the patient and the community.

FEIGNED INSANITY

Insanity is sometimes feigned, especially by those charged with crime or who have already been convicted. The object for so doing is apparent.

Mania, melancholia, and dementia are the **usual forms** simulated.

In examining such a case it must be remembered that each form of insanity presents a clinical history and symptoms, both mental and physical, peculiar to it, just as typhoid fever or any other disease does. Careful and repeated observation will usually enable the examiner to detect flaws in the simulated insanity. Thus, a patient who exhibits mental symptoms resembling **acute mania**, but who does not lose weight or who sleeps long periods spontaneously, would be fraudulent.

Or it would be equally suspicious if the mental symptoms of a person resemble **aggravated melancholia**, while food is taken, and the circulation, digestion, etc., are good.

A study of the **mental symptoms** may also be helpful. Thus, for instance, systematized delusions do not occur in mania or non-systematized ones in paranoia. Simulators usually exaggerate their symptoms, and hence frequently feign mental disturbances not belonging to the form of insanity simulated.

In all cases a decision should only be reached after close study, and, if necessary, repeated observation.

In this connection, it is well to know that convicts who most of the time are well behaved may have sudden attacks, in which they are destructive and noisy. These attacks last a few days and then pass off almost as suddenly as they came. There is apparently no desire to feign, and the subjects of the attack will tell you that they do not know why they did it, but they could not help it. Such attacks are familiar to all who are associated with the management of prisons (p. 551).

IDIOCY

Classification.—Idiocy has already been defined. In degree, these cases vary from children who are feeble-minded or backward (in other words, those who while capable of mental improvement and of being some use in the world are a few years behind their age in mental development) to those whose minds are practically blank. It must be understood that while the word "children" is here used an idiot who reaches adult age is still an idiot.

An adequate classification of idiots is difficult to make. That of Peterson is as follows:

1. Hydrocephalic idiocy.
2. Microcephalic idiocy.
3. Paralytic idiocy.
4. Epileptic idiocy.
5. Traumatic idiocy.
6. Sensorial idiocy.
7. Meningitic idiocy.
8. Myxedematous idiocy or cretinism.
9. Amaurotic idiocy.
10. Idiot savants.

Hydrocephalic, microcephalic, epileptic, traumatic, and meningitic idiocy are readily recognized by their symptoms and history, as is also the paralytic variety, which is the mental enfeeblement due to the lesions that also cause the so-called cerebral palsies of children (p. 249). Amaurotic idiocy and cretinism have already been described (pp. 256 and 515).

The term **idiot savants** has been applied to all idiots or imbeciles, from whatever cause, that exhibit special aptitudes of one kind or another, as in music, calculating, memorizing, special subjects, etc.

Sensorial idiocy is a form due to a congenital or early loss of two senses, as sight and hearing. Such may be educated to a high degree of mental development.

Space will not permit of a detailed description of all of these forms, and only the general points will be here described.

Some idiots have physiognomies resembling those of the lower races of man. These are often spoken of as *mongolian*, *negroid* idiots, etc.

Etiology.—Peterson classifies the cause of idiocy as follows:

Degenerative.	Hereditary transformation of nervous and mental diseases.		
	Pathological heredity in the form of vitiating diseases or habits (tuberculosis, rheumatism, gout, syphilis, alcoholism, etc.).		
	Sociological factors (extreme youth of parents, extreme age of parents, disproportionate age of parents, consanguinity).		
Adventitious.	Gestational.	Maternal.	Trauma, shock, fright, diseases, maternal impressions.
		Fetal disorders.	Syphilis, heart disease, arteritis, morbid processes in the brain and meninges, twin pregnancy.
	Parturitional.		Difficult labor, primogeniture, premature birth, asphyxia at birth, instrumental injuries, pressure on the cord.
	Postnatal.		Convulsions, cerebral diseases, trauma to the head, febrile diseases, mental shock, sunstroke, overpressure at school.

Symptoms.—Idiotic or imbecile children often develop, physically as well as mentally, more slowly than do normal children, and frequently the complaint of parents when the bring these children to the physician is that, while of the proper age, it has not learned to walk or is unable to sit up without support. Of course, in cases of paralytic idiocy this might be due to the paralysis; in the class of cases referred to, no evidence or history of paralysis can be found. Further examination will then usually elicit the facts that teething did not begin as soon as it should, and that its progress has not been normal, and that the child is small for its age. While this is a usual history, there are instances in which the physical development is as good, and sometimes better, than in normal children; and the failure of the child to develop mentally as it should first calls attention to it. Mentally it does not act as other children. Often such children are irritable and bad-tempered; they do not notice objects or people; attention cannot be attracted; and they do not show other well-known evidences of awakening intelligence. If the child is older, it will be found that it has either not learned to talk at all, or has been slow in doing so; that it will not feed itself, and that it continues to soil itself as infants do. Some of the stigmata of degeneration (p. 518) will be found, especially the high V-shaped hard palate.

After learning to walk, great restlessness and muscular activity are shown by many of these cases. They continually want to be on the go; are irritable, mischievous, and impatient of restraint. Others are quiet; will stay in one place for a long time playing with some trifling objects.

They may be destructive, cruel, and have homicidal instincts. Various forms of sexual perversion and masturbation may be present. Some cases have a tendency to steal, and show great cunning in securing coveted objects. When sent to school they learn slowly, if at all, and are several grades behind children of their own age.

Of course, all idiots do not present all of the symptoms mentioned above. There are various grades of intelligence and tractability, which, however, are not always synchronous. A child may be quiet and well-behaved, but is not able to learn satisfactorily. Othere may learn fairly well when they try, but are mischievous, irritable, or possibly criminal in their instincts. Others, again, may be especially bright in some one direction (*idiot savant*). Idiots, especially those that do not learn to talk, sometimes emit peculiar sounds. Peculiar muscular movements often occur, as rhythmic movements of the limbs, rotary movements, etc. These are independent of the various posthemiplegic movements (athetosis, choreiform movements, etc.) which may occur in cases of paralytic idiocy.

Diagnosis.—The diagnosis is based on the fact that the child either from birth does not or at some period during the developmental period ceases to progress mentally and ethically as it should. The type of the condition would be determined by the history and examination of the case. Thus the extremely small head would denote the microcephalic form; the development of the condition after the onset of cerebral paralysis the paralytic form, etc.

Upson[1] has reported cases of mental retardation apparently due to non-erupted and impacted teeth, determined by the *x*-rays, as the patients improved when the trouble was remedied.

[1] Rev. Neurol. and Psychiat., August, 1910, p. 457.

It must not be forgotten that idiots may sometimes develop some form of insanity, and also that they are often given to violent, almost maniacal outbursts of temper, which must not be confounded with true mania.

Prognosis.—Of course, these cases cannot be cured; but by proper means of education many of them can be made fairly useful members of society.

Treatment.—The only treatment is the sending of the child to an institution devoted to the education of such children; but if of such a type that nothing can be done there for him, committal to an asylum should be advised.

Accompanying conditions, as epilepsy or paralysis, should be treated as elsewhere advised.

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